

THE THERAPEUTIC CONTROL OF HYPERCHLORHYDRIA
ASSOCIATED WITH PEPTIC ULCERATION

By

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INTRODUCTION.

Man's original aims in life, so we are assured, were probably only three in number - to remain alive, to obtain food, and to propagate his species. For all practical purposes, these still represent his essential aims, though with the increasing complexity of civilisation, their realisation has become proportionately less simple. The necessity for food is probably the most primitive and immediate of these functions, as history bears witness; from Sulla's repeal of the corn grants and the banquets of Trimalchio through the ages to Napoleon's return from Moscow and the present economic stresses of Europe, Food has been the factor prompting Destiny.

"Food has its uses" - but, thanks to human nature, its abuses also: and it is with the results of its abuses that Medicine has been largely concerned throughout the centuries. With the rapidly increasing speed of existence now obtaining, the abuses and their effects are becoming daily more marked, and the resultant ill health encroaching more and more upon the time and therapeutic ingenuity of the physician.

"Stomach trouble" is almost universal: even the daily Press is filled with articles dealing with the subject and advertisements of so-called remedies for it. And in Medicine, there is a surprising lack

of unanimity as to the best approach to treatment:
quot homines, tot sententiae. The present thesis
has been undertaken in an attempt to assess the value
of present day treatment, and to consider the efficacy
of diet and drug in the treatment of "stomach trouble"
in one of its most puzzling guises - peptic ulcer-
ation and hyperchlorhydria.

This thesis is divided roughly into three
parts. The first part deals with the general
aspects of the problem, the current theories in
connection with it, and a consideration of the more
important published work on the subject. The
second part consists of a series of reports of
actual cases, and includes biochemical and radio-
logical details. In the third and final part, an
attempt is made to discuss the problems raised in
a more detailed manner, and with special reference
to the cases included in the thesis. A list of
references to authorities quoted is appended at
the end of the thesis.

THE THERAPEUTIC CONTROL OF HYPERCHLORHYDRIA
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INVESTIGATION OF GASTRIC FUNCTION.

From the earliest times the problems of digestion have interested medical men, but comparatively little of value has emerged from the early writings on the subject, although there were unrivalled opportunities for research in the earlier eras of civilisation. Reamur (1752) and Spallanzani (1783) were probably the earliest investigators to approach the question in a scientific manner. They introduced food in sponges into the stomachs of animals, studied the food after recovery, and recognised that digestion involved chemical processes. Prout (1824) and Tiedman and Gmelin (1824) showed independently that the acidity in gastric digestion was due to the presence of free hydrochloric acid. A few years later Beaumont (1833) reported his observations on a patient with a gastric fistula resulting from a gunshot wound. He studied this patient for eight years and was able to demonstrate the presence of other substances than hydrochloric acid in the gastric juice, these findings leading to the discovery of pepsin by Wassermann in 1839. Bidder and Schmidt (1852) showed that hydrochloric acid is produced by the gastric glands, and Heidenhain (1878) laid the foundations of the "surgical" investigation of digestion later elaborated by Pawlow (1897) and his pupils.

The method of study of gastric secretion by fractional analysis of specimens obtained by the stomach tube, is according to Maly, largely due to the initial

investigations by Leube (1871) and Kulz (1875). Although the tube has been used for centuries for feeding and lavage, it was not until this period that it was used for the study of gastric contents. For a long time after this, however, the value of the method was not appreciated until the stimulus to investigation was given by Ehrmann, Ehreulich and Ettinger in Germany, and Rehfuess and his associates in America. The tube used was stiff and of large calibre, and the introduction of the Eindhorn catheter was a decided improvement. Both this and the Ryle tube are of small calibre, and of great value for the "liquid" meal. This method has become widely used, and although supplemented recently by the alcohol and the histamine meals, is sufficiently accurate for most work.

GASTRIC SECRETION - ANIMALS.

(1) Invertebrates.

Among the invertebrates, no free acids or proteolytic ferments requiring free acids for their digestive action have ever been conclusively demonstrated. The acid reaction often present appears to be due to acid salts or acid saliva, though Griffith (1892) states that Fredericq noted hydrochloric acid in the gastric juice of Mya.

Bodansky and Rose (1922) demonstrated the presence of pepsin in certain coelenterates (Stromolophus; Physalia) active at pH3.0 which is approximately the

same as mammalian pepsin. Free mineral acid (sulphuric acid) in certain molluses is unaccompanied by ferments, and probably of different significance to mammalian hydrochloric acid. Free mineral acid is said to appear in the digestive vacuoles of some unicellular animals, but proteolytic action does not proceed till this acid is fixed or neutralised, and its function is considered to be less for digestive purposes than for killing ingested spores. It would appear that invertebrate digestion is essentially pancreatic and intestinal. (Von. Furth, Griffith, and Jordan).

(2) Fish.

The work of Sullivan (1907) and Biedermann (1911) is of considerable importance in this connection. Hydrochloric acid and pepsin appear to be secreted by the gastric glands in all fish, normally in the region of the gastric sac corresponding to the mammalian fundus. The presence of sulphuric acid and organic acids has also been reported. Secretion differs from mammalian secretion in that there is a much higher concentration of hydrochloric acid (Svolima, 1919); further, fish pepsin acts rapidly at low temperatures, and there is a high optimum acidity for action.

(3) Amphibians, Reptiles, Birds.

Hydrochloric acid and pepsins are present, and act in digestion, but to date comparatively little is known of processes involved. Nervous and hormonal control is

at present being investigated, but the results so far are inconclusive and conflicting.

4. Mammals.

It is in the mammalia that gastric secretion has been most intensively studied, and naturally enough a great deal has been in man himself. The gastric secretion in the higher mammals is remarkably alike, and this similarity has led to a considerable amount of experimental work in animals and application of the results to human beings.

It is recognised that in the normal mammal, secretion is controlled by both a nervous and a reflex mechanism (Clarke, 1937). The nervous control is probably exercised through the vagus, and it has been shown that the injection of atropine diminishes secretion. Hydrochloric acid is secreted by the cardiac and not by the pyloric portion of the stomach. Under normal conditions, secretion is started by a psychic reflex, which is supplemented by a chemical reflex which maintains secretion while food is in the stomach.

The latter reflex is considered to be due to a hormone (the "gastrin" of Edkin) from the pyloric mucosa. The gastric contents pass into the duodenum, while still slightly acid, and the pylorus closes until neutralisation is effected. In the presence of acid gastric contents, there is a tendency for regurgitation of alkaline duodenal contents to take place, this regurgitation probably varying with the degree of acidity. It is thought that the

stomach in man secretes about 1500 c.c. of gastric juice per day, in which there is $\frac{1}{2}\%$ hydrochloric acid, but considerable variation occurs.

Variations in the composition of relative values of constituents of the gastric juice have been noted in normal and pathological states. Variations in the hydrochloric acid content have received a great deal of attention, and it is with increase in this constituent (hyperchlorhydria) that the present thesis is concerned, though decrease (hypochlorhydria) or absence (achlorhydria) are not uncommon.

HYPERCHLORHYDRIA.

In view of the remarkable variations in the hydrochloric acid content of the gastric juice, it is difficult to decide when the acidity is excessive. Vanzant and his colleagues at the Mayo Clinic (1932), investigating a series of 3746 cases of apparently normal people, put the average normal upper limit of free acidity as between 35 and 45, while Bennett and Ryle consider the figure to be lower. (The figure used expresses the number of cubic centimetres of decinormal sodium hydroxide required to neutralise the free hydrochloric acid in 100 c.c. gastric juice). Much higher readings have been recorded in apparently normal people, however, and hyperchlorhydria per se cannot be regarded as pathological.

(a) Causes of Hyperchlorhydria.

Hyperchlorhydria is commonest during the "active period of life," and hence is most often found in persons between the ages of 20 and 40 years. Long working hours, irregular meals, nervous strain and worry all play an important part in the aetiology, and excessive smoking has also been implicated. Most commonly it is associated with peptic ulceration, and it is interesting to note that gastric ulceration, when present, occurs with equal frequency in both sexes, whereas duodenal ulceration is three to four times more common in men than women. It may, however,

give rise to no symptoms, and in some cases is a "pure neurosis" (Saville 1933).

It may occur as an accompaniment of other alimentary disturbance e.g. in colitis, appendicitis, cholecystitis, or cholelithiasis, or intrapelvic disease and disease in the female genital tract. There is some evidence to show that it may be familial. That it may be associated with hormonal imbalance has been shown by Dodds and Noble (1937) (pituitary) and Moore (1938) (thyreoid and pancreas), while Cushing (1932) has pointed out its occurrence in certain neoplastic cerebral conditions. Hurst has frequently emphasised the constitutional factor, or "hypersthenic diathesis."

(b) Physiology, and pathology of secretion.

Hydrochloric acid is formed by the parietal or oxyntic cells of the stomach from the blood chlorides, and three phases of digestion are involved in its production, namely, the psychic, the gastric, chemical or hormonal, and the intestinal.

Various hypotheses have been put forward in connection with the maintenance of acidity. Pavlov (1910) considers that the amount of secretion varies with the type of stimulus, although the acid content of the secreted juice is constant (about 0.5%), and that the juice undergoes dilution and neutralisation in the stomach. Rosemann (1911) considers that the acid content varies with the type of stimulus, while

the total chloride content of the juice is constant. It has been suggested by Boldyreff (1915) that the high initial acidity is reduced by regurgitation through the pylorus of alkaline fluid from the duodenum and the upper small intestine - a view which has received the support of many clinicians.

The Boldyreff hypotheses has received much consideration, especially by Bolton and his colleagues, (1927, 1928, 1931, 1933, 1936) and in their opinion is an important factor in maintaining normal acidity.

Further support has been afforded by the work of Portis and Portis on animals (1926), Elman (1929) on chemical grounds, and Wilhelmj (1926, 1937 a and b) from the biochemical point of view. Many workers, however, are in agreement with McCann (1929) and Goldberg (1932) who believe that the reduction of acidity is effected by an intragastric mechanism, by which there is a gradual decrease in the rate of acid secretion and a combination of the secreted acid with mucin from the prepyloric part of the stomach. Others suggest that the duodenal regurgitation acts by dilution of the gastric contents rather than by neutralisation.

It has been shown that the introduction of hydrochloric acid into the duodenum causes a relaxation of the pyloric sphincter and an inhibition of acid secretion in the stomach, and Gray (1937) pointed out that co-ordination of these two processes, set in

action by the entrance of acid chyme into the duodenum, may be an important factor in regulating the acid level in the stomach.

Recently the intragastric acid-regulating mechanism has come to be studied closely, and the part played by buffer substances investigated. The pyloric mucous membrane secretes fluid rich in mucus, and this substance has a strong power of acid neutralisation. It seems probable that a combination of the Boldyreff mechanism and the intragastric neutralising mechanism is responsible for preventing the acidity from becoming too high.

The modern outlook on the question has been discussed by Wilhelmj and his co-workers in a series of papers, and may be summarised briefly.

- (1) Regurgitation of non-acid fluids from the duodenum is a factor in the reduction of gastric acidity, their action being both neutralising and diluting.
- (2) Inhibition of acid secretion occurs when acidity rises beyond a certain point.
- (3) When acid passes into the intestine, there is an inhibition of gastric acid secretion.
- (4) The psychic factor may be powerful enough to prevent inhibition of secretion, even with the presence of acid in the stomach and intestine.
- (5) In some cases the pyloric part of the stomach, and in others vagal influence, may play a part in the control of acidity.

- (6) In some cases excessive absorption of water by the stomach may cause hyperchlorhydria.

Other factors may be involved in the production of hyperchlorhydria e.g. continuous secretion of acid, or unduly prolonged secretion. Faber (1935) has suggested that gastritis of the pyloric antrum may cause prolonged secretion, and that the more quickly the stomach emptied in such a case, the sooner it would contain almost pure juice and the higher acidity would rise.

He states that juxta-pyloric ulcer is frequently associated with an erosive gastritis in this region, and hyperchlorhydria might result from this mechanism. This type of gastritis, without the presence of ulceration, would have a similar effect. This pyloric gastritis or gastro-duodenitis may spread, and if involving a large area in the stomach, may cause hypochlorhydria or achlorhydria. Peptic ulcer on the gastric side of the pylorus is very rarely found, and pyloric ulcer is 99% duodenal. The marked association of gastritis and duodenal or "antral" ulcer has only been stressed by German observers; American and English workers have not found it, and consider that the multiple ulcers and gastritis are peculiar to Central Europe countries, though in this country Hurst has stressed gastritis as a precursor of ulcer.

If the Boldyreff hypothesis is considered, it can easily be seen that if the neuromuscular pyloro-

duodenal mechanism is disturbed, either by local or reflex cause, the regulation of gastric acidity may be profoundly affected. Local causes (e.g. aural irritation, ulcer) or reflex disturbance (e.g. by sensory nerves from chronic appendicitis) by interfering with this mechanism might cause failure of duodenal regurgitation, pylorospasm or increased gastric tonus, and hence hyperchlorhydria. If the intragastric mechanism for regulation of acidity is considered, it can be seen that this mechanism can similarly be affected by local or reflex causes, with resultant hyperchlorhydria.

It may be concluded that the concentration of free acid in the stomach at a given time, therefore, depends (Moore, 1938) on

- (1) the number of parietal cells secreting
- (2) the length of time during which the parietal cells respond to a stimulus by secreting e.g. if the mucous membrane is in a state of irritation, the duration of response may be prolonged above normal.
- (3) the degree of duodenal regurgitation.
- (4) the operation of the intragastric acid-neutralisation mechanism, of which at least part is the formation of alkaline fluid by the pyloric mucosa.

Physiologically, the fourth factor is possibly more important than the third in the control of acidity, but in pathological states it is probable that the first and second factors are of more significance than

the others, though all four are involved. The present state of knowledge in the correlation between physiological and pathological processes does not permit of more definite conclusions.

(c) Relation of Hyperchlorhydria to Peptic Ulcer.

It is probable that a high acidity renders the gastric and the duodenal mucosa liable to injury, and is a predisposing cause of peptic ulceration and of gastritis; further, its presence is thought to delay healing when ulceration has taken place.

Hurst has expressed the view that hyperchlorhydria associated with juxta-pyloric ulceration is almost certainly causal rather than resultant to the ulcer and should be considered as an expression of the hypersthenic gastric diathesis.

Experimentally, a great deal of work has been done in an attempt to elucidate the relationship between hyperchlorhydria and peptic ulcer. Mann and Bollmann (1932) carried out a series of experiments designed to diminish or eliminate the mechanism of neutralisation of the gastric contents on evacuation from the stomach, and were successful in producing intestinal ulcers. They demonstrated these ulcers formed at the point where the gastric contents impinged or were led over on discharge from the stomach. Ulcers were also produced by feeding dogs with hydrochloric acid over a period of time. From

their studies they concluded that the excess acid factor was of extreme significance. These observations have since been confirmed and extended by other workers. The original work which laid the foundations for this type of research were mainly by Mann (1925, 1927, 1929), and Ivy (1919, 1920, 1929); since then, a very large number of papers have appeared; but basically the procedures have not altered. It is easy, by numerous methods, to induce haemorrhagic erosions in the stomach; the real difficulty is to get a chronic ulcer. These small erosions heal readily (and it is possible that similar ones may occur in the human being) but it is difficult to obtain the factors which prevent healing.

Jejunal ulcers were produced by draining the duodenum into the ileum and then performing a gastio-jejunosomy; these ulcers did not form if the duodenal drained into the jejunum near the anastomotic site. (Mann and Williamson, 1923).

Simpson (1934) fed animals suffering from an induced gastritis with hydrochloric acid, and noted that they developed gastric erosions. Animals without gastritis did not develop erosions on a similar acid diet, and animals with gastritis did not develop erosions on an ordinary diet. The conclusions drawn were that hyperchlorhydria is a potentially dangerous state, and may be the forerunner of acute ulcer or

erosion in the presence of gastritis or an unhealthy gastric mucous membrane. Clinically, gastroscopy seems to support this view.

Other methods of producing ulceration experimentally include the administration of cinchophen to dogs, resulting in a diffuse haemorrhagic gastritis, and a rapid increase in the amount of acid juice secreted. In a few weeks these processes subside, leaving one or two ulcers which persist and become chronic. (Bollmann, Stalker and Mann 1937, 1938). There is an extraordinary parallelism in behaviour and structure between these ulcers induced in dogs by cinchophen and human peptic ulcer, findings which suggest that normally there is a state of balance between the digestive attack of acid gastric juice and the defences of the gastric mucosa. This balance is upset in the dog by a poison which damages the defences and at the same time strengthens the attack. This type of ulcer is probably the most useful yet available for experimental purposes.

It is worthy of note that the modern tendency is to regard peptic ulcer as of neurogenic origin - an idea originally put forward by Rokitansky and Virchow and since supported by Cushing (1932). Further support has been lent by Beattie (1932), and Watts and Fulton (1935) by their work on hypothalamic stimulation; by the work of Manning, Hall and

Banting (1937), on vagal stimulation: by Nechelles (1937) on acetylcholine; and by Babkin (1938) on the relation between vagal activity and histamine production. Dodds and Noble have pointed out the gastro-toxic effects of certain pituitary extracts, and stressed the close relationship between the pituitary and the brain with special reference to parasympathetic nuclei. Consideration of this, in association with the fact that the hypothalamus is thought to be the "seat of the emotions" seems to reconcile the prevalence of duodenal ulcer among the "lean, wiry, nervous, ambitious" type of person. Babkin sums up the problem thus: "Under certain circumstances, owing to some defect in the secretory mechanism, gastric secretion may deviate from its normal course, and conditions may arise which initiate destructive processes in the gastric mucosa."

It can be seen that peptic ulceration and hyperchlorhydria may readily give rise to a "vicious circle," the ulceration diminishing the pyloric reflex and so binding neutralisation, and so increasing the hyperchlorhydria and preventing healing of the ulcer. It is upon this assumption that the alkali and dietetic treatment of peptic ulcer associated with hyperchlorhydria is founded, though it is probable (Faber, 1935: Lindsay and Evans, 1929) that neutralisation of the hyperacidity is not the only factor involved. Smithies

(1937) considers that the establishment of "physiological rest" is of more importance than the neutralisation in promoting healing. It is generally accepted, however, that diminution or elimination of hyperchlorhydria plays a large part in treatment.

It has been suggested that hyperchlorhydria per se may be a factor in inducing gastritis of the pyloric antrum, with pylorospasm and increased tonus. It is obvious that in this case again there will result the establishment of a "vicious circle."

As regards the morbid anatomy of the condition: there may be no recognisable lesion associated with hyperchlorhydria, but in most cases there is peptic ulcer, antral gastritis, or duodenitis. The introduction of the gastroscope, however, has increased the possibilities for research in this field.

Davies, in his Bradshaw Lecture (1936) has pointed out that "although much is still obscure in the actiology of peptic ulcer as seen in man, experimentally it has been shown that a chronic ulcer can be readily produced, and that active gastric juice is by itself sufficient to produce this lesion in the intestine." This statement is of course, controversial, but seems at the moment to express fairly well the attitude adopted by most clinicians: in actual fact the results of research and the opinions of workers are so conflicting as to warrant the

inclusion of the questions of the aetiology of peptic ulcer and its relation to hyperchlorhydria with the astrophysical mathematics of Eddington, de Sitter and Lemaitre as "a summation of possible probabilities."

Treatment.

It may be of value to outline the general scheme of medical treatment adopted; the present thesis is not concerned with the surgical aspects of treatment.

"Rest in bed" is the first principle adopted. The patient is kept in bed for at least three, and in some cases for more than three, weeks, and is not allowed to get up until all pain, tenderness and muscular rigidity is absent. Dietetic measures are instituted, the diet being liquid first, easily digestible, and given as small frequent feeds (2-hourly in the early stages). Various special diets have been devised, and of these the most widely used are McLean's, Sippy's or Lenharz's or some modification of these; in the present instance, Sippy's was used (Appendix I.). In combination with these, an "acid neutralising" drug or group of drugs is administered, and in some cases atropine or belladonna. On general lines, stimulants of any description are forbidden, and the use of alcohol and tobacco discontinued. Any septic foci e.g. teeth or tonsils, receive due care, and a regular bowel movement is ensured. Such, in brief, are the therapeutic measures adopted.

PRESENT INVESTIGATION.(1) Scope.

In the preceding pages an attempt has been made to review the present day conception of the ulcer problem, and it will be seen that the essential underlying cause still eludes us. Hence we must adapt, improve and supplement our methods of treatment by application of physiological principles and careful clinical and laboratory study. In recent years it has become more obvious than ever that surgical measures, which cannot remove the underlying cause, have only a very limited scope in treatment, and the pendulum has swung back to "medical" forms of treatment. Under these conditions a greater responsibility falls upon the medical clinician to endeavour to improve methods of treatment, to get the quickest response, to safeguard against complications and to reduce the incidence of recurrence.

While the modern tendency is to regard ulcer as in some way an unfortunate side-effect of the nervous and emotional stresses to which the individual is exposed under present day conditions, and as such the result of factors over which the clinician cannot have complete control, yet the fact remains that more locally and specifically the control of the acid content of the stomach is one of the prime factors in promoting the rapid healing of a peptic ulcer.

We have seen that ulcers will not form in the stomach or duodenum unless free acid is present. Surgical measures are only successful in so far as they reduce hyperacidity.

To control acidity is to remove the biggest immediate factor which is preventing healing, and it was with the idea of arriving at some conclusion as to how far this can be accomplished, and what effect therapeutic measures have upon the acidity of the stomach, that the present investigation was undertaken.

(2) Methods Employed.

Forty-four cases were investigated, divided into the following groups:-

- (1) Cases of hyperchlorhydria and peptic ulcer on full Sippy-alkali regimen.
- (2) Cases of hyperchlorhydria and peptic ulcer on full Sippy regimen with Magsorbent in place of alkali.
- (3) Cases of hyperchlorhydria and peptic ulcer on light diet alone, without the exhibition of any drugs.
- (4) A series of normal cases as controls on diets corresponding to those of Group 1, 2 and 3, as above.
- (5) A series of cases of hyperchlorhydria and peptic ulceration on which laparatomy was performed, and diagnosis and conclusions either confirmed or denied.

Full details of the Sippy regime will be found in Appendix I. of this thesis. The alkalis used were

Mag. oxid. lev grs. xxx) Powder
Sod. citras grs. xxx) No. 1.

and

Calc. carb. grs. xxx) Powder
Sod. bicarb. grs. xx) No. 2.

On admission to hospital, a careful history was obtained from each case, and a complete physical examination carried out. This was followed by a radiological examination and any other investigation thought necessary e.g. blood examination, estimation of basal metabolic rate, etc. The Wassermann reaction was determined, and the stools tested by the benzidine method for the presence of occult blood. An ordinary "gruel" fractional test meal was then carried out, and the presence or absence of hyperchlorhydria determined.

The Sippy or other treatment was then commenced, and on the first or second days following the institution of this treatment, the Ryle's tube was passed again, and a specimen of gastric contents obtained every hour for a period of twelve hours, the specimens being withdrawn midway between the taking of food or drug. This 12-hour meal (hereafter referred to as the long test meal) was repeated at a later date, usually at the end of the second, or during the third, week. In all cases, the pH. of the urine was determined each morning and evening by the use of the B.D.H.

Universal Indicator; the urine was also tested in the routine side-room manner. In the cases having alkalies during treatment, the blood area and the CO₂ combining power of the blood (Van ^{Slyke}~~Syke~~ method) was estimated every few days.

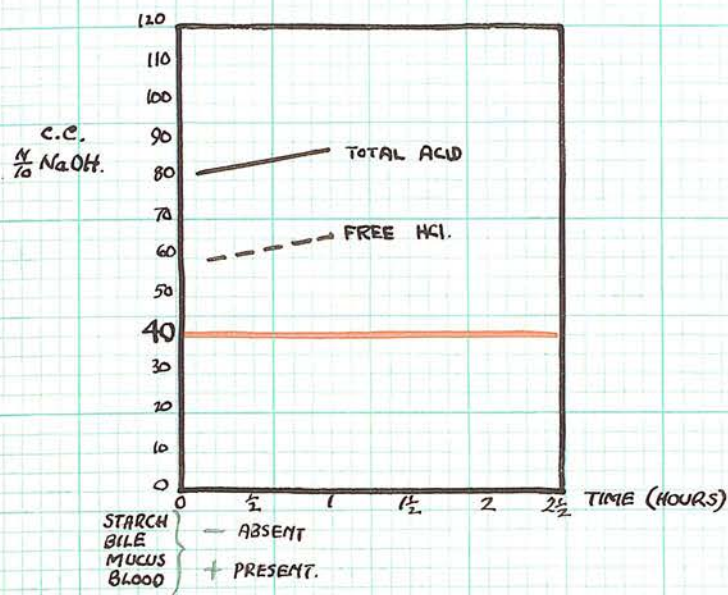
The specimens of gastric contents were obtained in the usual manner, employing a Ryle's duodenal tube. During the long test meal, this tube was left in situ for the whole period, but a few patients preferred to re-swallow it every hour, and this was permitted when desired. The free and total acid contents of the specimens were obtained by titration against decinormal sodium hydroxide solution, the indicators used being Topfer's reagent (dimethyl-amino-azo-benzol) and phenol-phthalein, and the results were noted in the form of graphs.

These graphs have been included after each case record, and a specimen page giving full explanatory details of the notation employed will be found immediately preceding the case records.

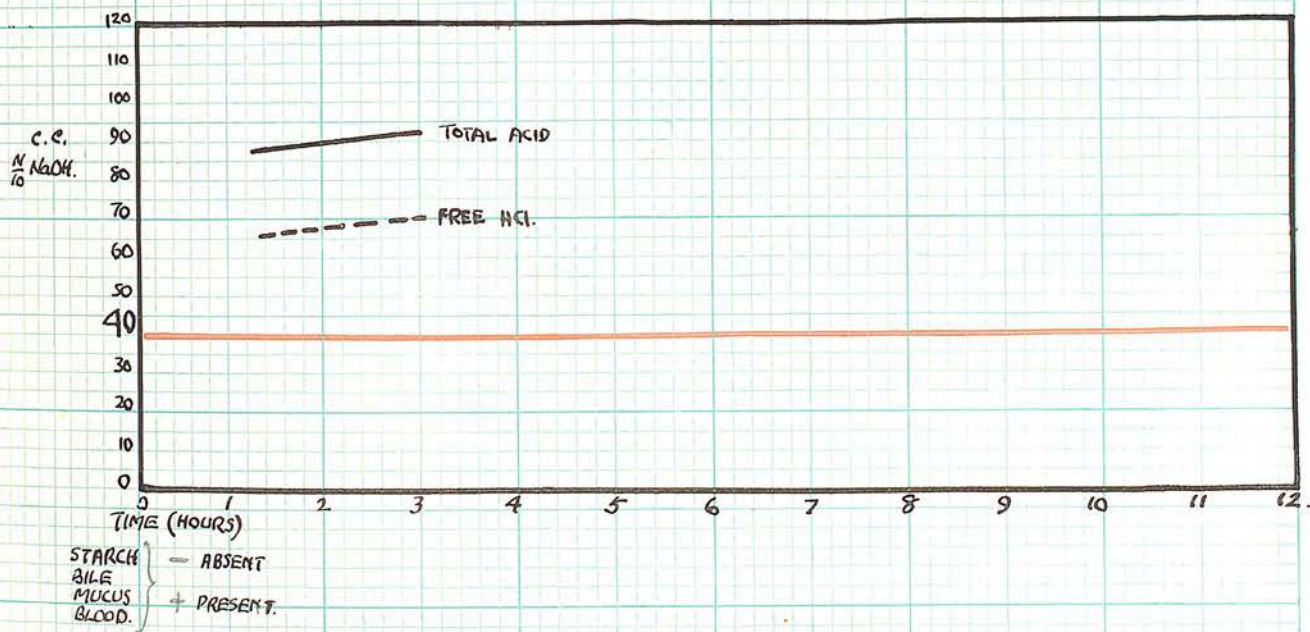
The notation employed is indicated in the following table:-

Series 1	=	light diet (control: series X).
Series 2	=	Sippy-Alkali (control: series Y).
Series 3	=	Sippy-Magsorbent (control: series Z).
Series 4	=	"Surgical."

SPECIMEN PAGE.



■ = MEAL.
■ = ALKALI OR MAGSORBENT.



Case (1) A.

Thomas McC. (aet. 36). Tram Conductor.

Admitted: 9:4:38.

Discharged: 16:5:38.

Complaint: Epigastric pain for two years.

Diagnosis: Duodenal ulcer.

Treatment: Light diet: no alkalis.

History: For about two years he has suffered from epigastric pain, gnawing in character and felt in the left and central epigastrium 2-3 hours after food. Flatulence is usually present, and eructation relieves pain, as does taking of food. Alkalis were tried about eighteen months ago, but were of no value and have not been used since. Appetite is good, but he is afraid to eat, particularly meat. About nine weeks ago, he had an attack of vomiting, bilious in character, and lasting throughout the day. It did not relieve the pain. Bowels move irregularly; and he has lost a lot of weight recently, about $3\frac{1}{2}$ stones in one year. The symptoms show definite remissions with rest and in the summer months.

Examination: He is a tall "gaunt" man, with no obvious osseous abnormality.

He smokes 10-15 cigarettes per day, and is a teetotaller and unmarried.

Alimentary: Tongue moist and clean; throat clear; there are a few carious teeth in the upper jaw.

Abdomen thin, moves well on respiration. There is

slight tenderness on palpation in the midline in the epigastrium, but no rigidity. Splashing not elicited. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 125/75.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach and oesophagus normal. Duodenal cap deformed and niche seen in first part - duodenal ulcer."

X-Ray of chest shows "Slight general increase of lung tissue markings throughout. Hila normal, show slight calcareous deposits. Translucency of lung tissue generally - emphysema."

Sugar tolerance test:

Urine.
trace.

Fasting blood sugar 113 mgm. %

$\frac{1}{2}$ hour after 50 gm. glucose 117 mgm. % nil.

1 hour after 50 gm. glucose 134 mgm. % nil.

$1\frac{1}{2}$ hours after 50 gm. glucose 124 mgm. % nil.

2 hours after 50 gm. glucose 120 mgm. % nil.

$2\frac{1}{2}$ hours after 50 gm. glucose 119 mgm. % nil.

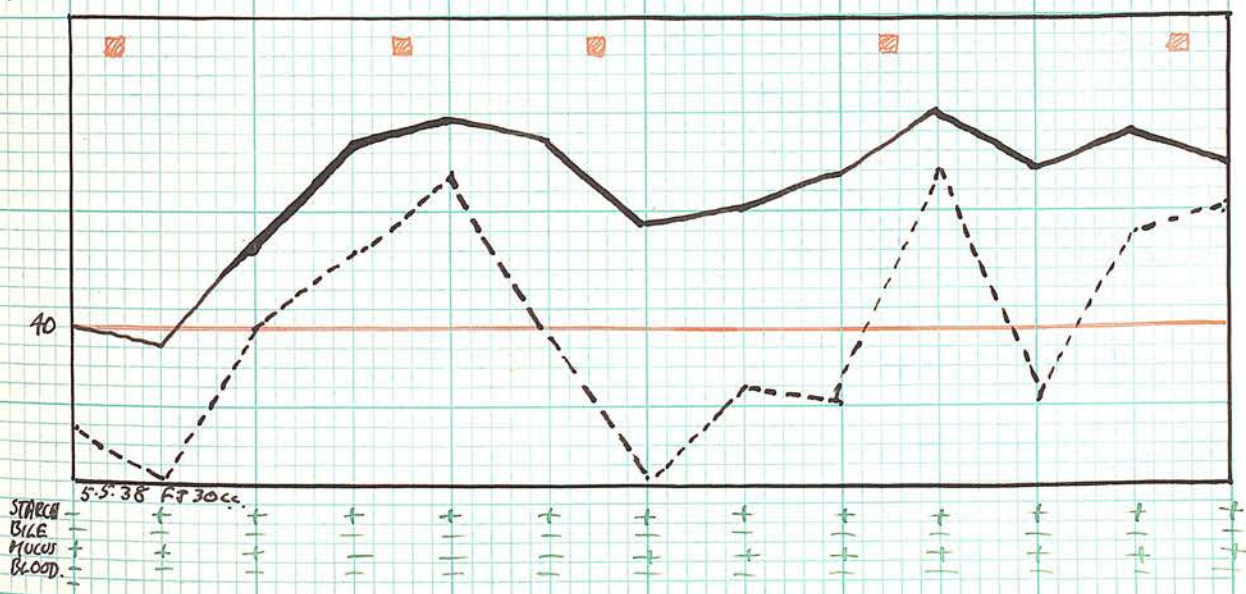
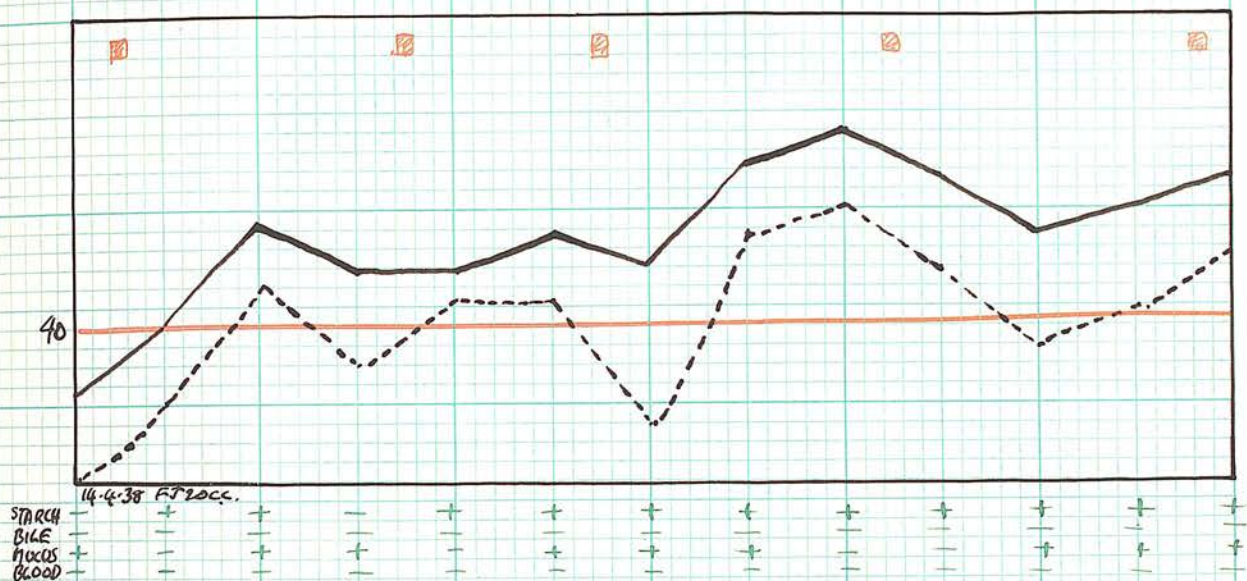
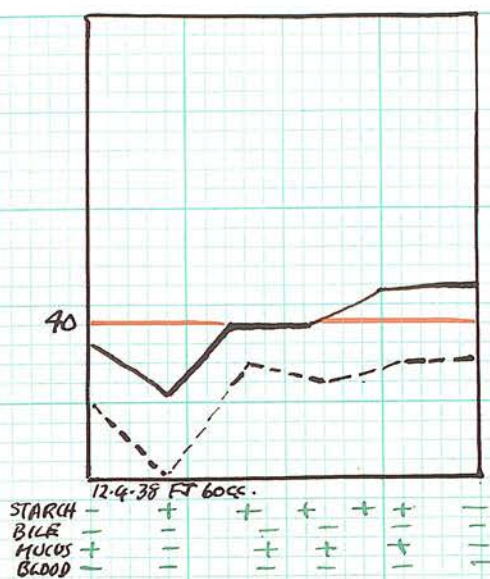
Eye examination VAR 6/6 VAL 6/6 (Snellen) Fundi and fields normal.

Urine negative for casts, albumen, etc. (done daily).
Urinary pH varied between 6.5 and 7.5 during his stay in hospital.

Notes: This man's general configuration suggested a mild acromegaly, but this was not confirmed. He had a duodenal ulcer, confirmed radiologically, and his short test meal showed no definite hyperchlorhydria. Long test meals, however, before and after treatment, showed a high degree of hyperchlorhydria, with prolonged spells during which the acidity exceeded 40. The second long meal was done two weeks after the first, and showed no noticeable improvement, although clinically he was well and symptom free. There has been no recurrence of symptoms within four months of discharge.

Conclusions: Clinically well; hyperacidity if anything worse as judged by test meal. Not controlled.

I. A.



Case (1) B.

Peter McC. (aet. 59). Tinsmith.

Admitted: 16:4:38. Discharged: 25:5:38.

Complaint: Epigastric pain for four years.

Diagnosis: Gastric ulcer.

Treatment: Light diet: no alkalis.

History: About four years ago patient began to have pain and discomfort in the epigastrium two hours after food. The pain sometimes passes through to the back, and is very acute and sharp, and is relieved by the taking of food. Recently it has wakened him every night about 1 a.m. It is always worse during the winter. In October 1937 he took a fainting turn and felt very weak and breathless, and for the following three days his stools were very black and hard. The pain has been much worse since. He has had two similar attacks since, accompanied by pallor, weakness and the passage of black stools, and on the last occasion he noted unchanged blood in the stools. He has lost weight recently.

Examination: He is a small pale man with an anxious expression. No osseous abnormality.

He smokes a pipe (3 oz. tobacco per week) and drinks spirits at the week-end.

Alimentary: Tongue dirty: teeth false: throat clear.

Abdomen moves well on respiration. There is an area to the right of the epigastrium which is tender on

palpation, but there is no muscular rigidity. No splashing was elicited. Liver and spleen not enlarged.

The descending colon can be palpated.

Cardiovascular: Nil to note. BP 90/55.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach J-shaped, good tone, peristalsis and motility. An incisura is present at the greater curvature side of pyloric antrum and a niche is seen at the lesser curvature. The duodenal cap is large and deformed. "Gastric Ulcer."

Barium enema - Nil abnormal detected.

Blood: RBC 3,800,000 WBC 9,400 Hb 70%.

Urine negative for casts, albumen etc. (done daily).

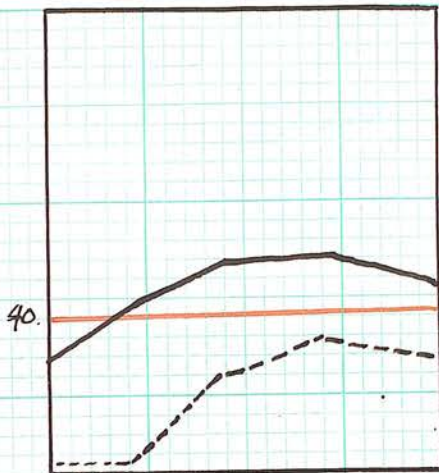
Urinary pH Varied between 6.5 and 7.5.

Notes: This patient is suffering from a gastric ulcer, confirmed radiologically, and probably chronic, as he has a history of previous haematemesis, and pain for a long period. Symptoms disappeared, and tenderness vanished within three days of admission, and there has been no recurrence four months after discharge. Test meal showed a very mild hyperchlorhydria, and a curve of the "climbing" type. A long test meal confirmed

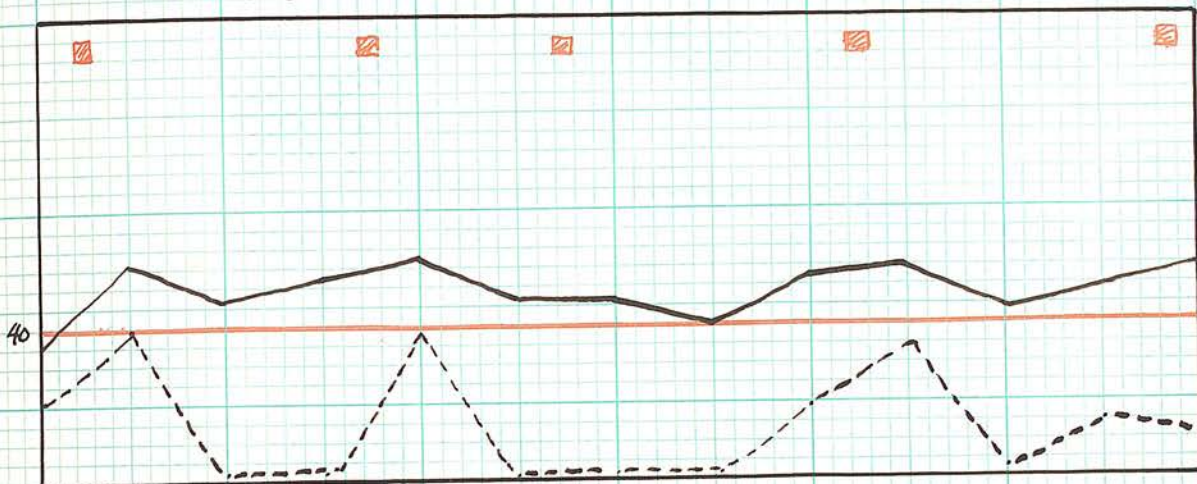
this finding, and showed a rise to 40 three times during the day. Another long test meal two weeks later showed a higher degree of acidity than previously, in spite of the fact that clinically he was well.

Conclusions: Clinically well: as judged by test meal, hyperchlorhydria is to all intents and purposes unaffected.

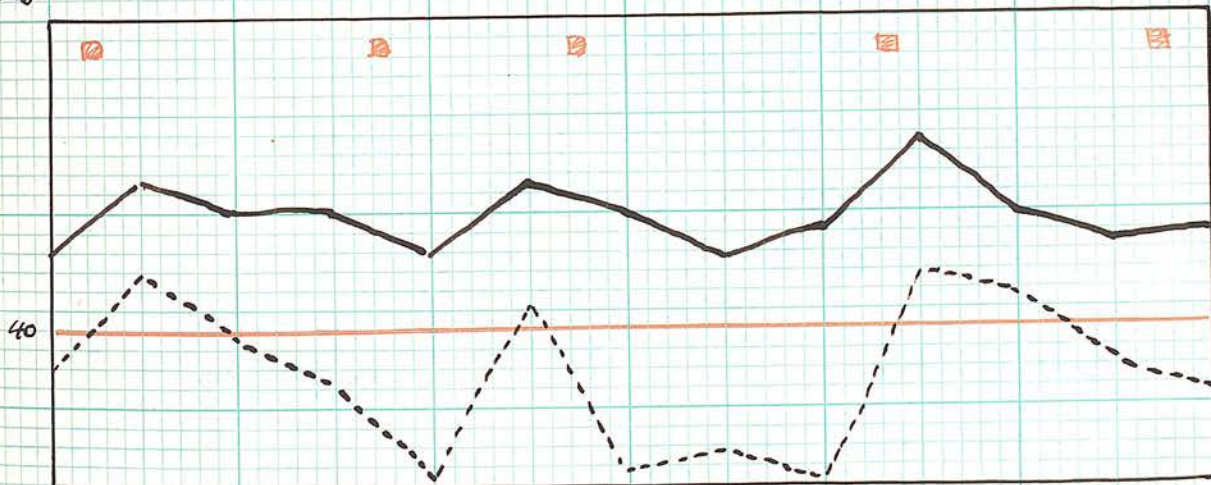
I.B.



	5-5:38	FT	30cc.
STARCH	+	+	+
BILE	-	-	-
MUCUS	+	+	+
BLOOD	-	-	-



	6.5.38	FT 30CC
STARCH	—	+
BILE	—	—
MUCUS	+	+
BLOOD	—	—



	30.5.38	RT	20cc
STARLIGHT	-	+	+
BILE	-	-	-
MUCUS	+	+	+
BLOOD	-	-	-

Case (1) C.

James L. (aet. 37). Insurance Agent.

Admitted: 29:3:38. Discharged: 29:4:38.

Complaint: Epigastric pain for eleven years.

Diagnosis: Duodenal ulcer.

Treatment: Light diet: no alkalis.

History: For the past eleven years he has suffered intermittently from attacks of epigastric pain, coming on about $1\frac{1}{2}$ - $2\frac{1}{2}$ hours after food, and not relieved by food. The pain is "stabbing" in character and occasionally felt in the back. He is also troubled with flatulence and acid eructations. He was in hospital three years ago following a haematemesis, but has not dieted since. Bowels are costive: he has not lost weight.

Examination: He is pale and drawn in appearance.

No osseous deformity.

He smokes about 15-20 cigarettes daily and drinks 1 pint of beer daily and occasionally whisky.

Alimentary: Tongue dirty: teeth good: throat clear, Abdomen is thin, but moves well on respiration. No tenderness or splashing is elicited, but the upper right rectus shows definite boarding. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 130/94.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative. (Done every second day).

X-Ray shows "Stomach and oesophagus normal. Slight degree of pylorospasm and duodenal cap much deformed and showing presence of an ulcer crater."

Blood: R.B.C. 3,800,000 WBC 10,600. Hb 76%

Urine negative for casts, albumen etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

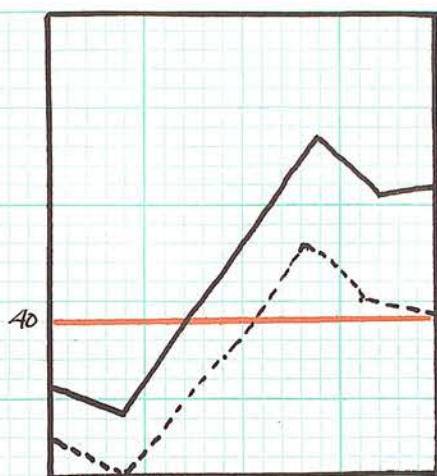
Notes: This man gives a long history of gastric trouble and has also suffered from an haematemesis. Radiologically there is an active duodenal ulcer, and considerable scarring suggestive of previous trouble.

His symptoms improved rapidly in hospital and he was clinically well on discharge and five months later.

His short test meal shows a severe degree of hyperchlorhydria and a "climbing" curve. This is confirmed in the long meals, which demonstrate a severe degree of hyperchlorhydria, to all intents unaffected by treatment, in spite of the symptomatic improvement.

Conclusions: Clinically well: as judged by test meal, severe hyperchlorhydria, unaffected by treatment. Not controlled.

I.c.

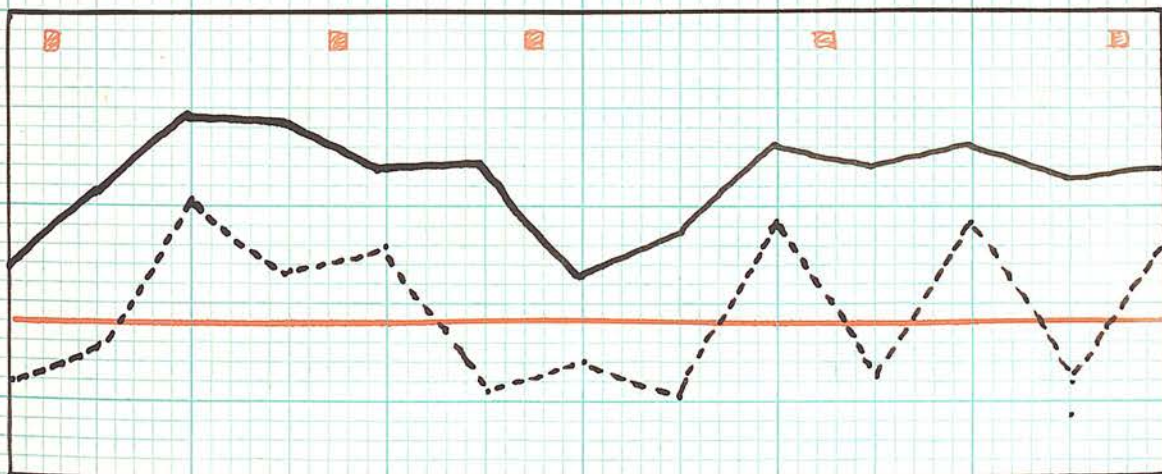


21.4.38. FJ30cc.

	24-45% N502	24-45% N502	24-45% N502	24-45% N502	24-45% N502
STARCH	+	+	+	+	-
BILE	+	-	-	-	-
MUCOS	-	+	+	-	+
BLOOD	-	-	-	-	-



22.4.38 FJ 40cc

[illegible]

7.5.38 (T) 30 cc

[illegible]

Case (1) D.

Susan G. (aet. 48).

Housewife.

Admitted: 18:4:36.

Discharged: 22:5:38.

Complaint: Epigastric pain for six months.

Diagnosis: Duodenal ulcer.

Treatment: Light diet: no alkalis.

History: For the past six months she has suffered from intermittent attacks of pain in the epigastrium, usually accompanied by bouts of vomiting. The pain occurs when she is hungry and is relieved by the taking of food or alkali. It is sharp in character and occasionally felt in the back. She has not lost weight. Bowels are regular: menstrual periods stopped three years ago. The pain wakes her sometimes at night - usually about 1 a.m. and subsides when she takes a drink of milk.

Examination: She is a slim woman, with rather a drawn look in her face, and shows no obvious osseous abnormality.

She is teetotal and does not smoke.

Alimentary: Teeth false, tongue clean, throat clear, abdomen is well covered and moves freely. There is slight tenderness in the midline in the epigastrium and some rigidity of the upper right rectus.

Splashing is not elicited. The liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 142/100.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative. (done every second day).

X-Ray shows "Stomach and oesophagus normal. Duodenal cap is persistently deformed and a niche seen." Urine negative for casts, albumen etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

Notes: This woman gives a short history of gastric trouble and is suffering from a duodenal ulcer, confirmed radiologically. Her symptoms subsided rapidly in hospital and rigidity and tenderness were gone after four days. Improvement has been maintained in the four months following her discharge. Her short test meal shows hyperchlorhydria associated with a "climbing" curve, and both her long meals confirm this and demonstrate that there has been no improvement in the degree of acidity to compare with the symptomatic improvement.

Conclusions: Clinically well: as judged by test meal, hyperchlorhydria unaffected by treatment.
Not controlled.

	2-4-50	2-5-50	2-6-50	2-7-50	2-8-50	2-9-50
STARCH	+	+	+	+	+	+
BILE	+	+	-	-	-	-
MUCUS	+	+	+	-	-	-
BLOOD	-	-	-	-	-	-



28-4-38 FJ 10cc

[illegible]

8.5.38 FT10cc.

[illegible]

Case (1) E.

Duncan R. (aet. 26). Carter.

Admitted: 4:4:38. Discharged: 5:5:38.

Complaint: Epigastric pain for three years.

Diagnosis: Duodenal ulcer.

Treatment: Light diet: no alkalis.

History: For the last three years he has suffered intermittently from attacks of epigastric pain, sometimes being free for as long as three months. In the last few months, however, the pain has become more severe and almost continuous. It is sharp in character, sometimes felt in the back, and comes on 1-1½ hours after food, and is relieved by food or alkali. There has been no vomiting, though he has sometimes felt nauseated. He has not lost weight. Bowels are constipated. Flatulence is troublesome, but there has been no acid eructation or heartburn.

Examination: He is a well built young man, and shows no sign of loss of weight. No osseous abnormality.

He smokes 10-15 cigarettes daily, and occasionally drinks beer - never spirits.

Alimentary: Tongue dirty: teeth good: throat clear.

Abdomen is well covered and moves freely. There is slight tenderness in the right epigastric region, but no muscle rigidity. Splashing is not elicited. Liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 120/90.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach and oesophagus normal. Duodenal cap deformed and niche seen - duodenal ulcer."

Urine negative for casts, albumen. etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

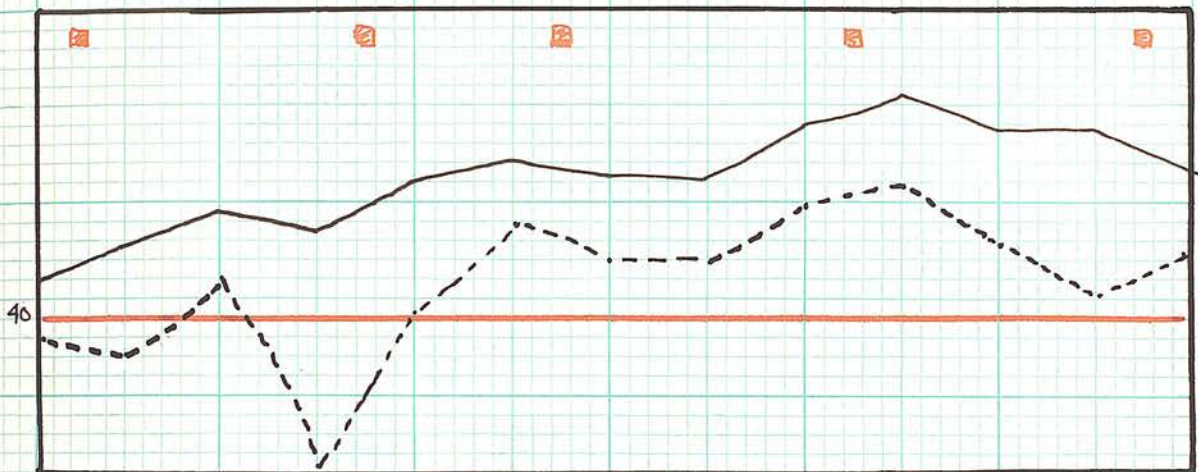
Notes: This man is suffering from a duodenal ulcer, confirmed radiologically. Rapid improvement took place in hospital, and within six days he was symptom free, and there was no rigidity or tenderness present. There has been no relapse in the four months since discharge. Test meals showed a definite hyperchlorhydria, and the two long test meals confirm this, and show no specific improvement following treatment.

Conclusions: Clinically well: as judged by test meal, hyperchlorhydria present and unaffected by treatment. Not controlled.

I.E.



5:5:38 FT 80 cc.
 S. + + + + - -
 Bl. + + - - - -
 M. + + - - - -
 Bl. - - - - - -



7:5:38 FT 80 cc.
 STARCH + + + + + + + + + +
 BLUE - - - - - - - - - -
 MUCUS + + + + + + + + + +
 BLOOD - - - - - - - - - -



10:5:38 FT 66 cc.
 STARCH + + + + + + + + + +
 BLUE - - - - - - - - - -
 MUCUS + + + + + + + + + +
 BLOOD - - - - - - - - - -

Case (2) A.

William W. (aet. 38).

Miner.

Admitted: 13:2:38.

Discharged: 7:3:38.

Complaint: Epigastric pain for three years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: For the past three years he has suffered off and on from attacks of epigastric pain, sometimes being free of it for as long as two months. Lately, however, it has been constant. The pain is burning in character, comes on about 1 $\frac{1}{2}$ -2 hours after meals, and is relieved by the taking of food or alkali. It is felt in the epigastrium to the right of the midline and does not go through to the back. In the last three weeks he has vomited several times and felt relieved afterwards. It is not influenced by different types of food, and does not wake him at night. He also suffers from flatulence, and heartburn. Bowels regular: he has never noticed any difference in the appearance of the motions. He has lost weight. No dietetic measures were adopted.

Examination: He is a thin, spare man with hollow cheeks and a rather anxious expression. No obvious abnormalities noted.

He smokes a pipe, using about 2 oz. black tobacco per week. Occasionally drinks beer, but has not done so for the last six months.

Alimentary: Teeth false, tongue dirty but moist, throat clear. Abdomen thin, moves well with respiration. Slight tenderness at right edge of rectus muscle, between rectus and costal margin. No muscle rigidity. Splashing easily elicited. Liver and spleen not enlarged. No pain, tenderness or rigidity elsewhere.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows ulder deformity of duodenum without evidence of pyloric stenosis.

Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power and urea (see graphs).

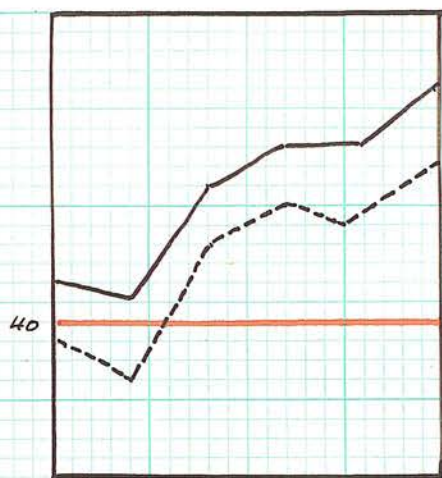
Urinary pH varied between 6.5 and 7.5.

Notes: This man is suffering from a duodenal ulcer, confirmed by X-Ray. Radiologically there is no pylorospasm, but clinical facts suggest that it is present. He became symptom free in three days in hospital, but tenderness disappeared on the fifth day. There has been no relapse six months after dismissal.

His test meal shows definite hyperchlorhydria, and a "climbing" curve. This is modified on the commencement of treatment, and the acidity falls slightly. This improvement is much more pronounced after two weeks, when the acidity (with the exception

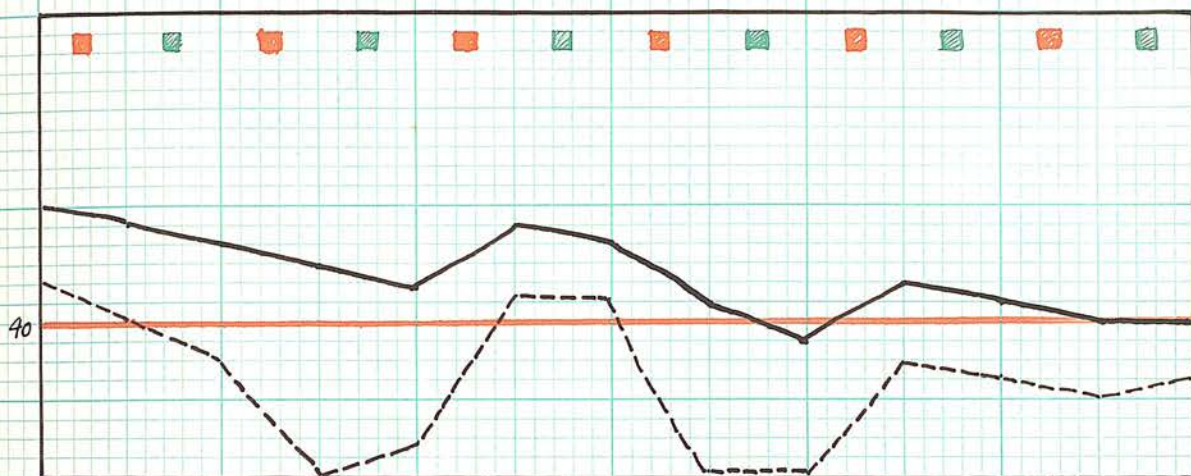
of that fasting juice) is well below 40 during the whole day.

Conclusions: Clinically well: hyperchlorhydria profoundly affected by treatment. No alkalosis developed during treatment. Controlled.



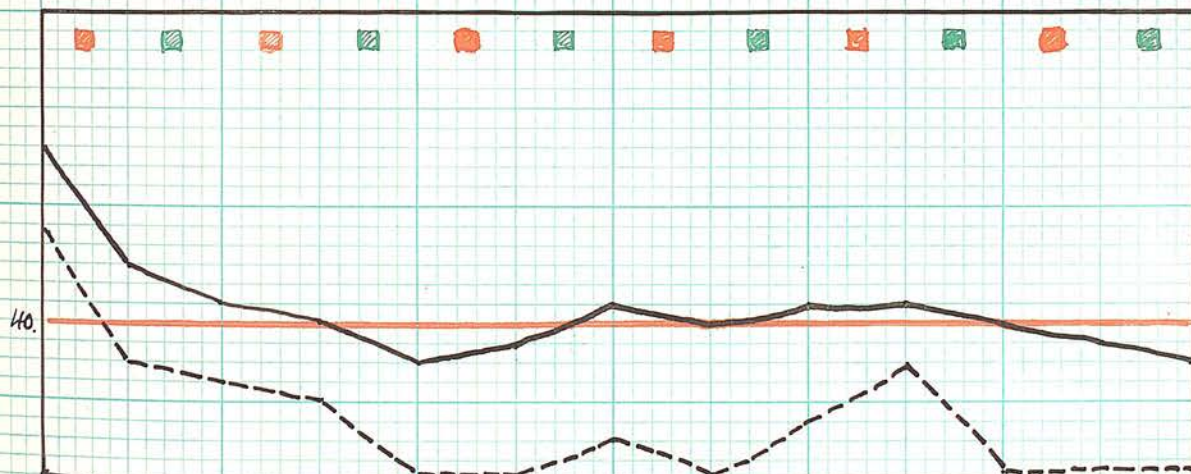
22-1-38 FJ 20 cc.

STARCH	+	+	+	+	+	+
BILE	+	+	+	+	+	+
MUCUS	+	+	+	+	+	+
BLOOD	-	-	-	-	-	-



25.1.38 FJ 3045.

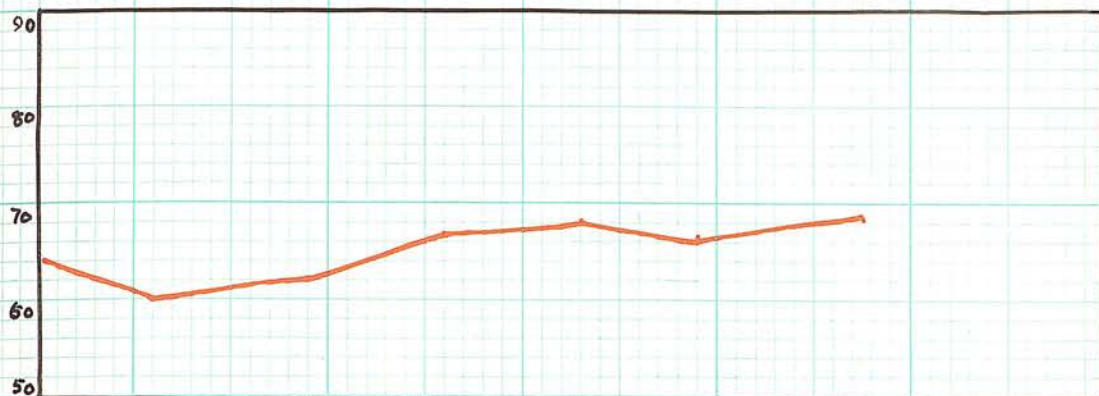
STARCH	+	+	+	+	+	+	+	+	+
SILE	+	+	+	+	+	+	+	+	+
MUUS	+	+	+	+	+	+	+	+	+
BLOOD	+	+	+	+	+	+	+	+	+



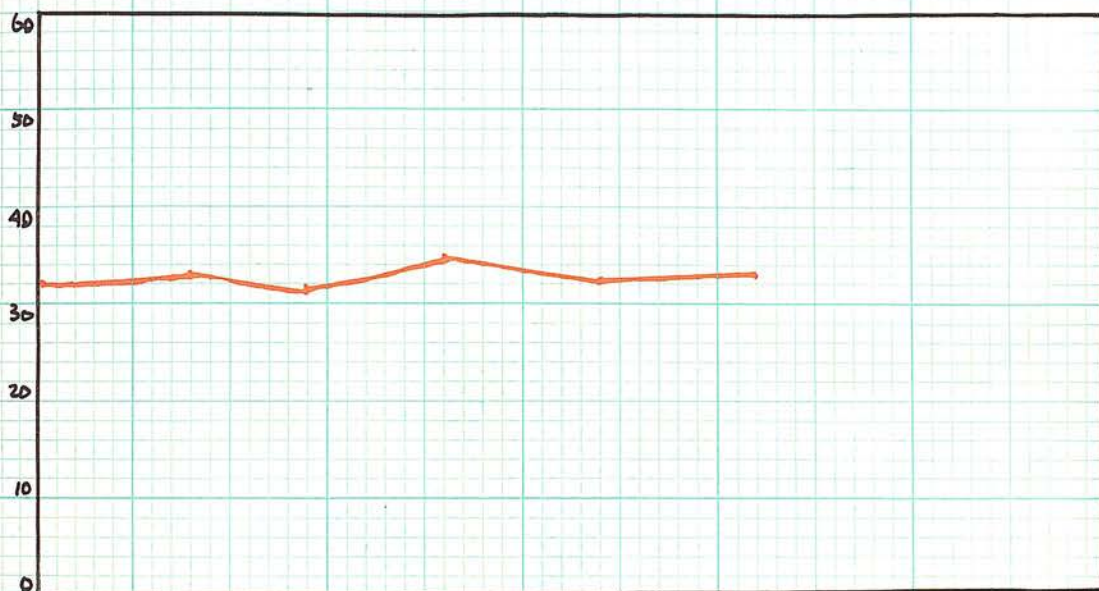
10.2.38 FT20CC.

[illegible]

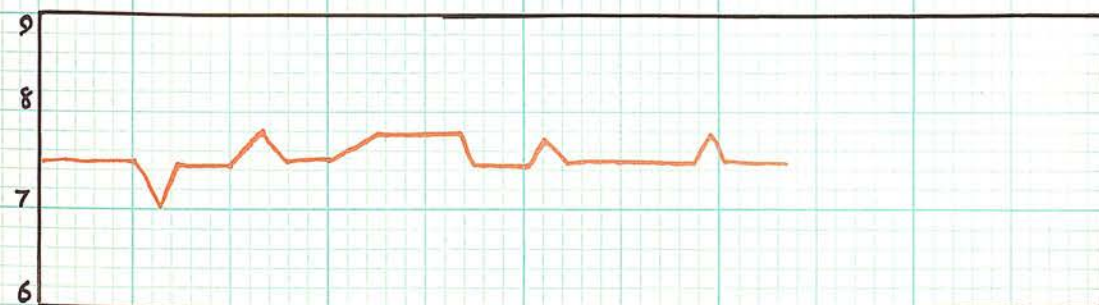
DAYS: 0 3 6 9 12 15 18 21 24 27 30 33



BLOOD CO₂ combining power. (Vol. %)



BLOOD UREA (mg. Hg. %)



URINARY pH.

Case (2) B.

Michael R. (aet. 42). Tramwayman.

Admitted: 7:2:38. Discharged: 6:3:38.

Complaint: Epigastric pain for twelve years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: For the past twelve years he has been troubled off and on with "indigestion" and "heart-burn." During the last year the abdominal discomfort has become worse and for the last nine months he has had almost constant epigastric pain, coming on worst about one and one half hours after food, and only slightly relieved by powders or food. Recently he has vomited once or twice - green stained undigested food - and this relieved him. His appetite is poor, and he has lost weight. He suffers considerably from eructations and flatulence, and the bringing up of wind eases the pain. Bowels tend to be costive, but he has not noticed any difference in the motions.

Examination: He is small, rather thin man with a "drawn" expression. No obvious abnormalities.

Smokes 10-15 cigarettes daily. Teetotaller.

Meals very irregular.

Alimentary: Top teeth false, bottom set carious.

Tongue furred and moist. Throat clear. Abdomen thin, but moves well on respiration. Slight muscular rigidity in upper right rectus, with

tenderness on deep pressure. No enlargement of liver or spleen. No splashing elicited.

Cardiovascular: Nil to note. BP 140/90.

Respiratory: Chronic cough. Occasional rhonchi heard in chest.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows scarring and deformity of duodenum with no evidence of gastric retention.

Urine negative for casts, albumen etc. (done daily).

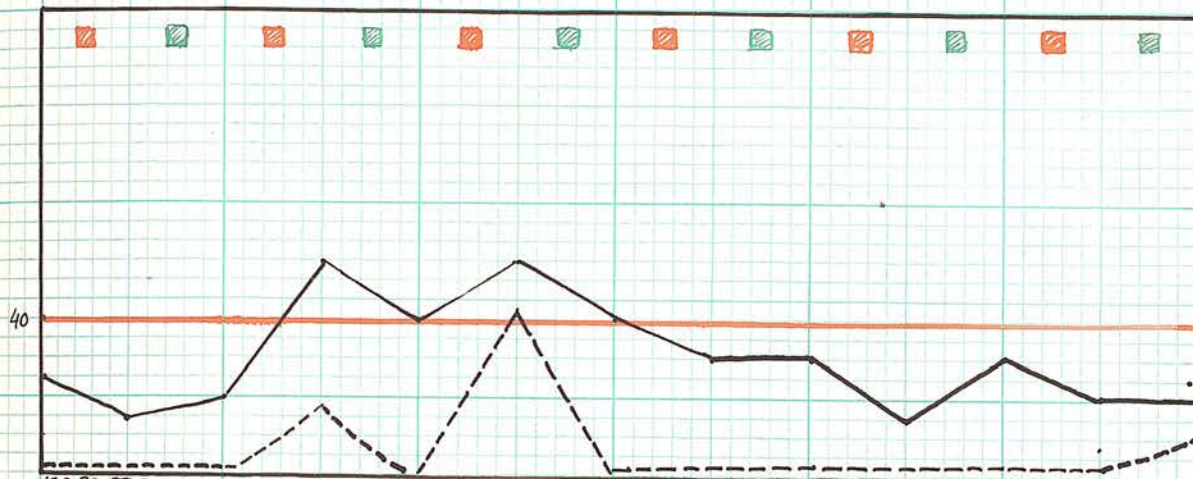
Blood CO₂ combining power and urea (see graphs).

Urinary pH (see graphs).

Notes: This man gives a long history of stomach trouble, and this, in conjunction with clinical and radiological findings, suggests the presence of chronic duodenal ulceration. Rapid symptomatic improvement took place in hospital, and there has been no relapse six months after discharge. Test meal shows a moderate degree of hyperchlorhydria: this is very markedly modified by treatment, as will be seen by comparing the long meals during treatment with the short one done before the institution of treatment. After two weeks, there is a further decrease in the acidity.

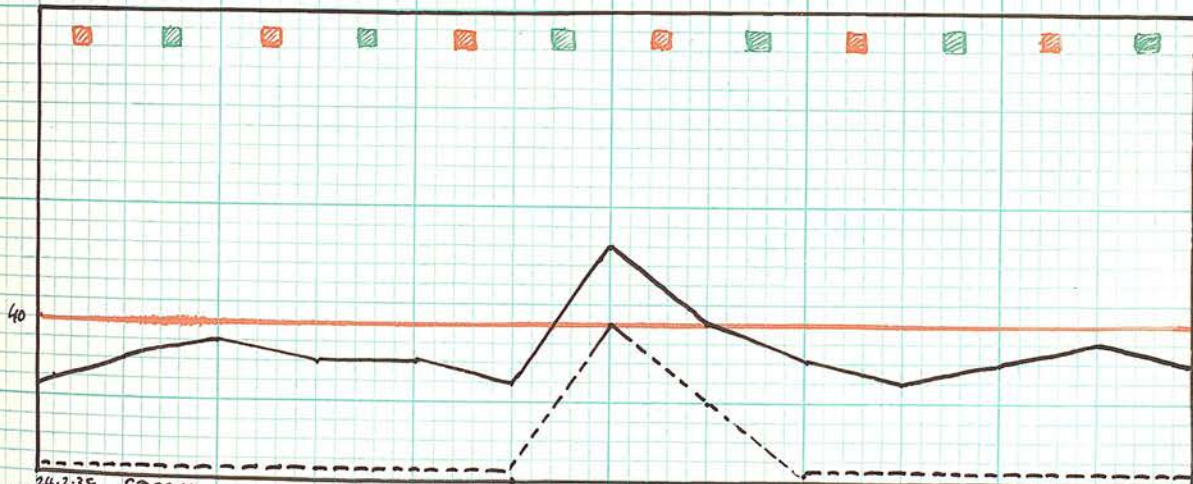
Conclusions: Clinically well: hyperchlorhydria
profiundly affected by treatment. No alkalosis.
Controlled.

STARCH	-	+	+	+	+	-
BILE	+	+	-	-	-	-
MUCUS	+	+	+	+	+	+
BLOOD	-	-	-	-	-	-



10.2.38 FT 30 c.

	10/25/88	11/30/88	12/30/88	1/30/89	2/28/89	3/30/89	4/30/89	5/30/89	6/30/89	7/30/89	8/30/89	9/30/89	10/30/89	11/30/89	12/30/89	1/30/90	2/28/90	3/30/90	4/30/90	5/30/90	6/30/90	7/30/90	8/30/90	9/30/90	10/30/90	11/30/90	12/30/90	1/30/91	2/28/91	3/30/91	4/30/91	5/30/91	6/30/91	7/30/91	8/30/91	9/30/91	10/30/91	11/30/91	12/30/91	1/30/92	2/28/92	3/30/92	4/30/92	5/30/92	6/30/92	7/30/92	8/30/92	9/30/92	10/30/92	11/30/92	12/30/92	1/30/93	2/28/93	3/30/93	4/30/93	5/30/93	6/30/93	7/30/93	8/30/93	9/30/93	10/30/93	11/30/93	12/30/93	1/30/94	2/28/94	3/30/94	4/30/94	5/30/94	6/30/94	7/30/94	8/30/94	9/30/94	10/30/94	11/30/94	12/30/94	1/30/95	2/28/95	3/30/95	4/30/95	5/30/95	6/30/95	7/30/95	8/30/95	9/30/95	10/30/95	11/30/95	12/30/95	1/30/96	2/28/96	3/30/96	4/30/96	5/30/96	6/30/96	7/30/96	8/30/96	9/30/96	10/30/96	11/30/96	12/30/96	1/30/97	2/28/97	3/30/97	4/30/97	5/30/97	6/30/97	7/30/97	8/30/97	9/30/97	10/30/97	11/30/97	12/30/97	1/30/98	2/28/98	3/30/98	4/30/98	5/30/98	6/30/98	7/30/98	8/30/98	9/30/98	10/30/98	11/30/98	12/30/98	1/30/99	2/28/99	3/30/99	4/30/99	5/30/99	6/30/99	7/30/99	8/30/99	9/30/99	10/30/99	11/30/99	12/30/99	1/30/00	2/28/00	3/30/00	4/30/00	5/30/00	6/30/00	7/30/00	8/30/00	9/30/00	10/30/00	11/30/00	12/30/00	1/30/01	2/28/01	3/30/01	4/30/01	5/30/01	6/30/01	7/30/01	8/30/01	9/30/01	10/30/01	11/30/01	12/30/01	1/30/02	2/28/02	3/30/02	4/30/02	5/30/02	6/30/02	7/30/02	8/30/02	9/30/02	10/30/02	11/30/02	12/30/02	1/30/03	2/28/03	3/30/03	4/30/03	5/30/03	6/30/03	7/30/03	8/30/03	9/30/03	10/30/03	11/30/03	12/30/03	1/30/04	2/28/04	3/30/04	4/30/04	5/30/04	6/30/04	7/30/04	8/30/04	9/30/04	10/30/04	11/30/04	12/30/04	1/30/05	2/28/05	3/30/05	4/30/05	5/30/05	6/30/05	7/30/05	8/30/05	9/30/05	10/30/05	11/30/05	12/30/05	1/30/06	2/28/06	3/30/06	4/30/06	5/30/06	6/30/06	7/30/06	8/30/06	9/30/06	10/30/06	11/30/06	12/30/06	1/30/07	2/28/07	3/30/07	4/30/07	5/30/07	6/30/07	7/30/07	8/30/07	9/30/07	10/30/07	11/30/07	12/30/07	1/30/08	2/28/08	3/30/08	4/30/08	5/30/08	6/30/08	7/30/08	8/30/08	9/30/08	10/30/08	11/30/08	12/30/08	1/30/09	2/28/09	3/30/09	4/30/09	5/30/09	6/30/09	7/30/09	8/30/09	9/30/09	10/30/09	11/30/09	12/30/09	1/30/10	2/28/10	3/30/10	4/30/10	5/30/10	6/30/10	7/30/10	8/30/10	9/30/10	10/30/10	11/30/10	12/30/10	1/30/11	2/28/11	3/30/11	4/30/11	5/30/11	6/30/11	7/30/11	8/30/11	9/30/11	10/30/11	11/30/11	12/30/11	1/30/12	2/28/12	3/30/12	4/30/12	5/30/12	6/30/12	7/30/12	8/30/12	9/30/12	10/30/12	11/30/12	12/30/12	1/30/13	2/28/13	3/30/13	4/30/13	5/30/13	6/30/13	7/30/13	8/30/13	9/30/13	10/30/13	11/30/13	12/30/13	1/30/14	2/28/14	3/30/14	4/30/14	5/30/14</
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24.2.38 FT 30 cc.

[illegible]

Case (2) C.

William M. (aet. 48). Railway Worker.

Admitted: 12:2:38. Discharged: 9:4:38.

Complaint: Epigastric pain for three years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: For the past three years he has been troubled by a feeling of discomfort and heaviness in the epigastrium, which has recently become actually painful. It comes on $1\frac{1}{2}$ -2 hours after food and is relieved by the taking of food or alkali. It is gnawing in character, felt in the midline, and sometimes in the back, and sometimes it wakes him at night. There has been no vomiting, but he suffers considerably from flatulence and heartburn. Bowels are constipated, and he has been losing weight.

Examination: He is a thin man, and looks much older than he really is. There is no osseous abnormality.

He smokes a pipe (2-3 ozs. tobacco per week) and is a teetotaller. He is married and has a good home.

Alimentary: Teeth false: tongue dirty: throat clear.

Abdomen is thin and moves freely on respiration. There is a tenderness on palpation in the right epigastric region, and definite muscular rigidity is apparent.

Splashing can be elicited. Liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 140/110.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach and oesophagus normal. Duodenal cap persistently deformed and niche visible - ulcer present."

Urine negative for casts, albumen etc. (done daily).

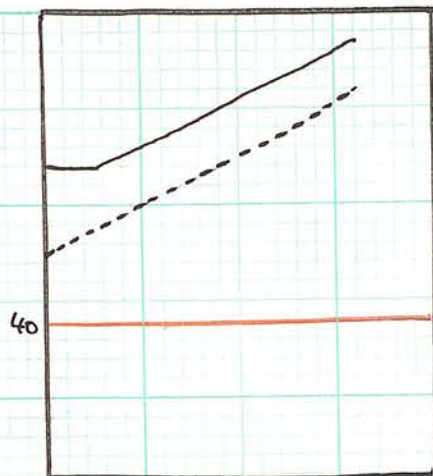
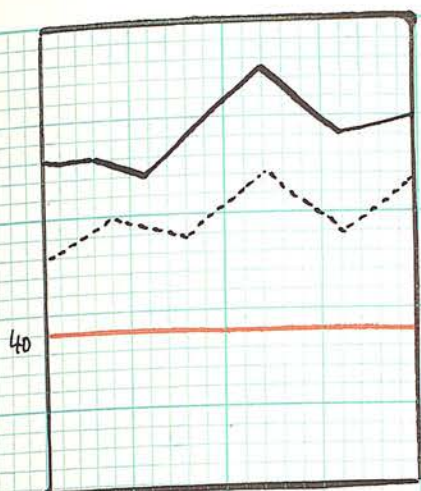
Blood CO₂ combining power and urea. (See graphs).

Urinary pH. (See graphs).

Notes: This man is suffering from the presence of a duodenal ulcer, confirmed radiologically. He improved rapidly in hospital and pain, tenderness and rigidity disappeared, but he never looked really well. He was well on discharge, and had a slight recurrence, lasting a few days, three months after discharge. He has been free for three months after this. Test meal shows severe hyperchlorhydria: long test meal shows that this is profoundly influenced by treatment, but that perfect control is not attained, as acidity exceeds 40 for a spell of several hours, in spite of therapy.

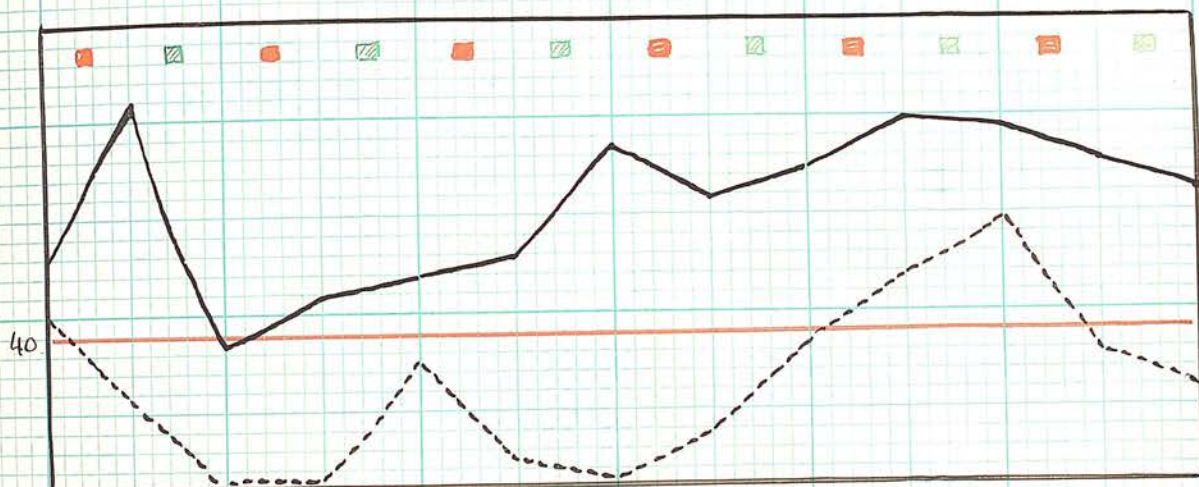
Conclusions: Clinically well, after mild relapse: hyperchlorhydria markedly modified by treatment: partially controlled.

2.c.



6.9.37 FJ 30cc.
 STARCH +
 BILE +
 MUCUS +
 BLOOD -

14.1.38 FJ 40cc.
 STARCH +
 BILE +
 MUCUS +
 BLOOD -



2.2.38 FJ 30cc.
 STARCH +
 BILE +
 MUCUS +
 BLOOD -

DAYS.

3

6

9

12

15

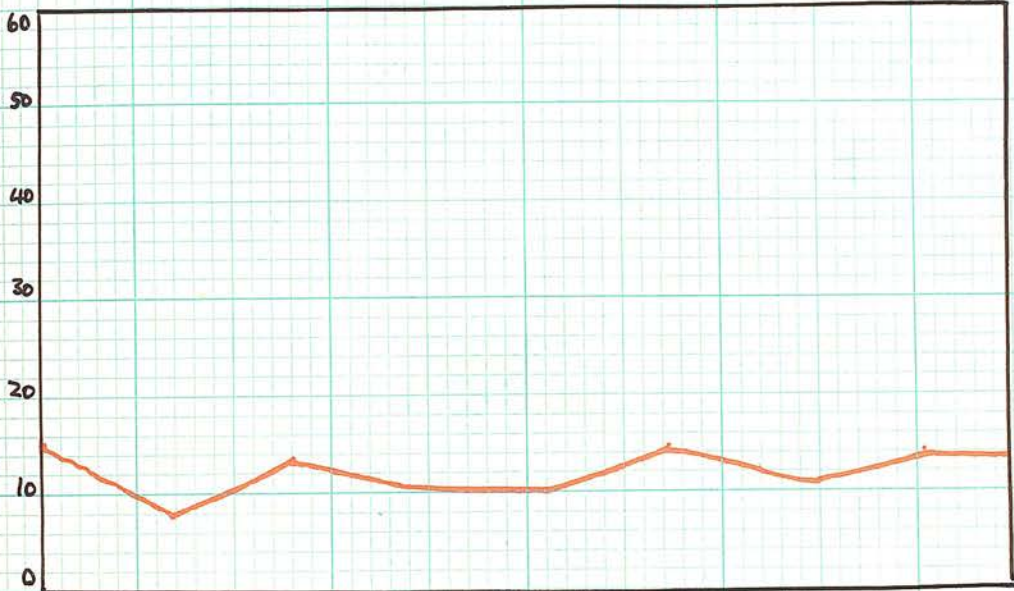
18

21

24

27

30

Blood CO₂ combining power (Vol-%)

Blood Urea (mgm-%)



Urinary pH.

Case (2) D.

Alex. M. (aet. 52). Miner.

Admitted: 13:1:38. Discharged: 28:2:38.

Complaint: Epigastric pain for six years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: For the past six years he has had epigastric pain, relieved by alkalis, curd vaguely related to food. It got worse and culminated one year ago in his vomiting a quantity of blood; he was admitted to hospital and was in for six weeks. On discharge he was very well, except for occasional dizzy turns and cough. About one week ago the pain returned, it was "jagging" in nature, and did not go through to the back: relieved by alkali but not food. It woke him at night, and during the day troubled him at irregular times. No vomiting. No loss of weight. No flatulence or heart-burn. No alteration in motions.

Examination: He is fairly well nourished, but does not look well. He is a slightly built man, with no obvious osseous abnormality.

Alimentary: Teeth false: tongue dirty: throat clear. Abdomen thin, moves well on respiration. There is slight rigidity of the upper right rectus on palpation, but no tenderness; he feels a twinge of pain on the examining hand being lifted. No splashing elicited. No enlargement of liver or spleen.

Cardiovascular: Nil to note. BP 115/80.

Respiratory: Chest is rather barrel-shaped. Percussion note is hyper-resonant; breath sounds are broncho-vesicular and expiration prolonged. Rhonchi are heard in all areas.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach and oesophagus negative. Duodenal cap is small and difficult to fill, and shows no evidence of stenosis. Ulcer was not demonstrated.

Electrocardiogram was negative.

Urine negative for casts, albumen etc. (done daily).

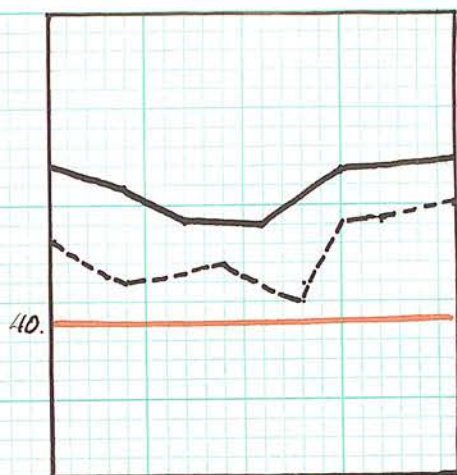
Blood CO₂ combining power and urea (see graphs).

Urinary pH. (see graphs).

Blood - Hb. 88%. R.B.C. 4,800,000 WBC 9,200.

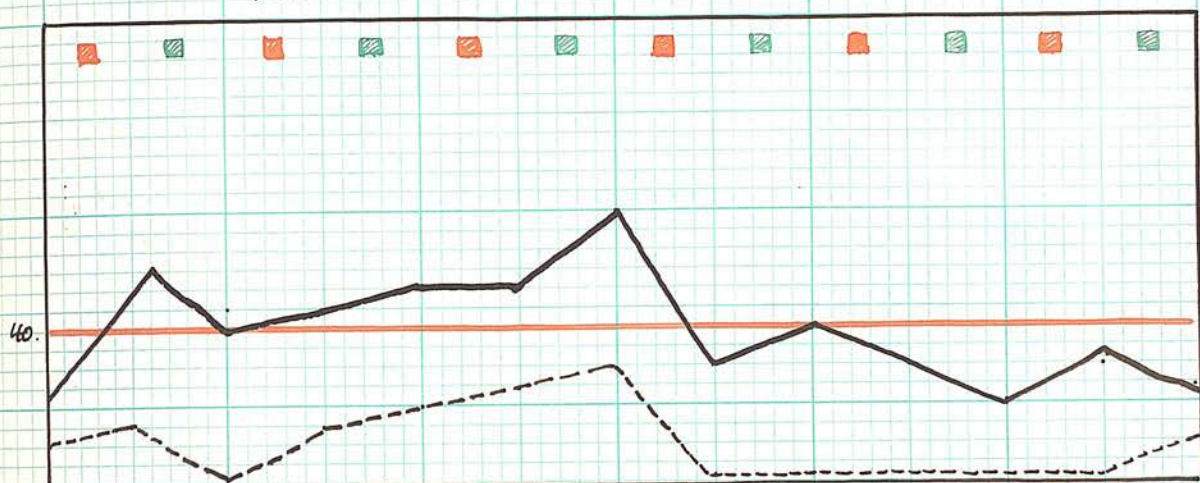
Notes: This man is suffering from the presence of a duodenal ulcer (not confirmed radiologically). Rapid improvement took place in hospital and there has been no relapse since discharge eight months ago. Test meal shows fairly severe hyperchlorhydria: this is markedly modified by the institution of treatment, and even further modified after two weeks.

Conclusions: Clinically well: hyperchlorhydria modified by treatment. No alkalosis. Controlled.

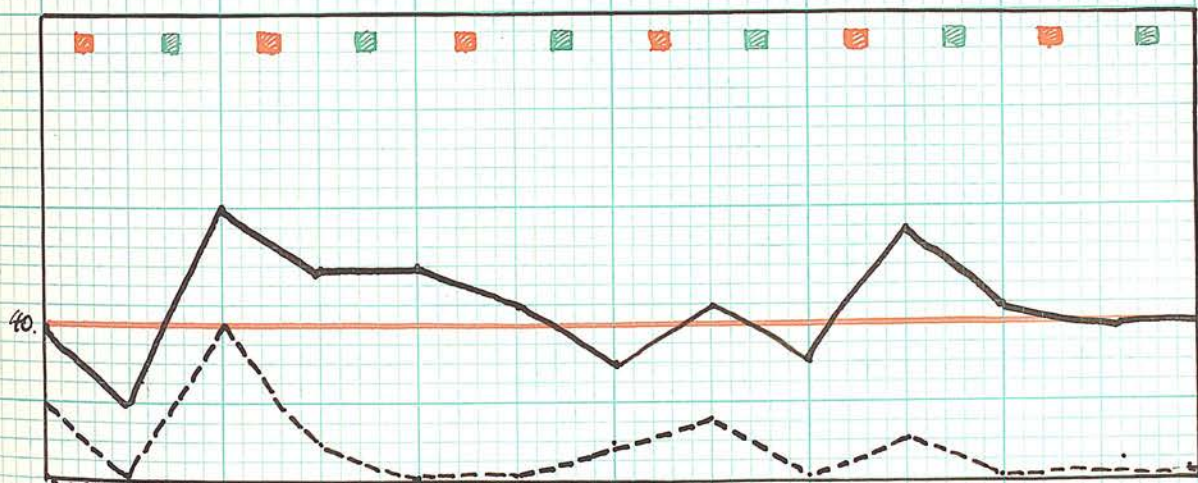


17.1.38 ФТНОС.

STARCH	-	+	+	+	-	-
BILE	-	-	-	-	-	-
GLUC.	+	+	+	-	+	+
BLOOD.	-	-	-	-	-	-



18-1-38 FJ 40cc.

[illegible]

32.38 FJ40 cc.

[illegible]

DAYS.

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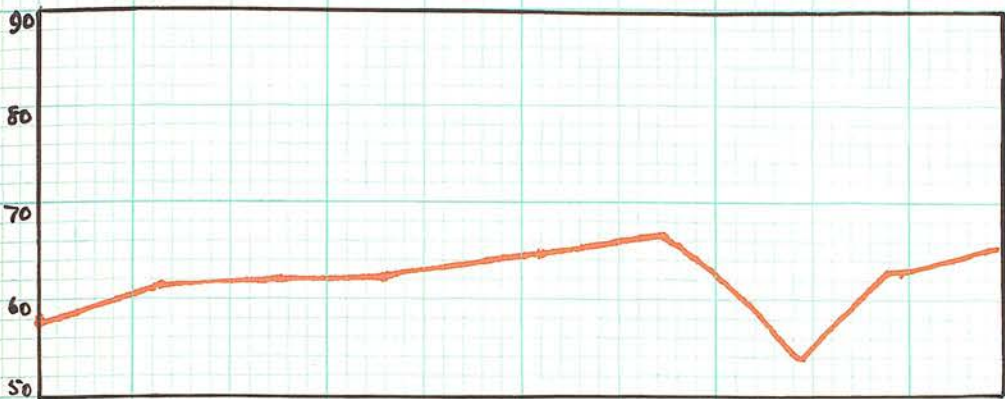
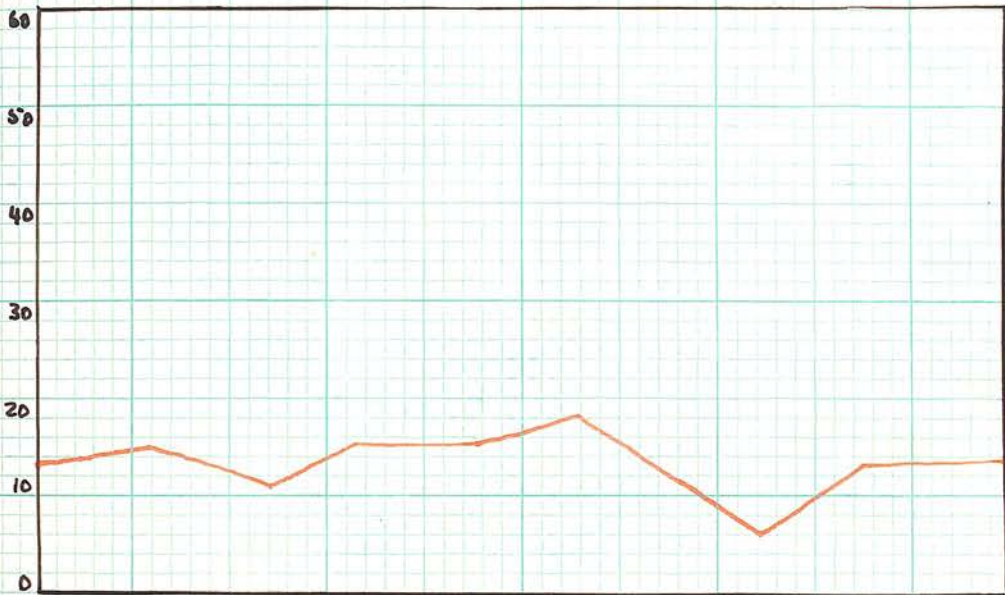
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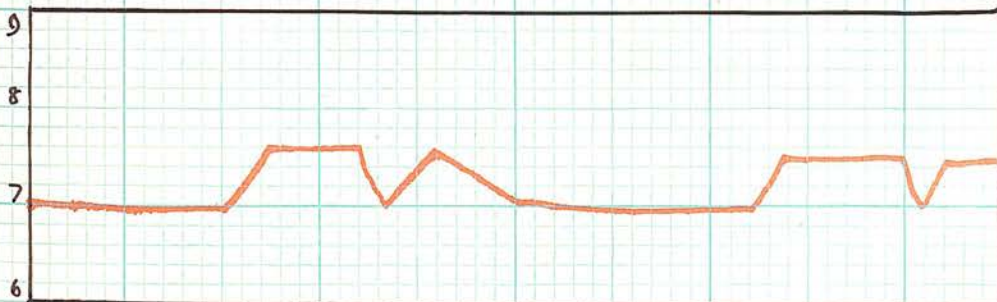
24

27

30

Blood CO₂ combining power (Vol%)

Blood urea (mgm. %)



Urinary pH.

Case (2) E.

Mungo S. (aet. 27). Electrical Linesman.

Admitted: 13:1:38. Discharged: 24:2:38.

Complaint: Epigastric pain for six years.

Diagnosis: Pylorospasm secondary to congenital duodenal deformity.

Treatment: Sippy-alkali.

History: For six years he has suffered from intermittent epigastric pain. It is "like a knife," and is greatly relieved by meals, alkalis and by self-induced vomiting. Attacks are accompanied by sour eructations and waterbrash, and last for periods varying from a few days to 3-4 months, with spells of freedom between. The free spells may be as long as three months. There has been very little vomiting, and no loss of weight. The pain is felt in the "pit of the stomach," and does not move elsewhere. It comes on during the day, between meals, and never at night. Bowels are constipated.

Examination: He is a strong, healthy looking young man, but in spite of this has rather an apprehensive expression. No osseous deformity.

He smokes 20 cigarettes per day, and drinks 2-3 pints of beer at week-ends. He is unmarried, and has good lodgings.

Alimentary: Teeth good: tongue furred: throat clear.

Abdomen well covered, moves well with respiration.

There is some tenderness in the right upper quadrant

of the epigastrium, but no muscle rigidity. Splashing elicited fairly easily. The liver and spleen are not enlarged.

Cardiovascular: Nil to note BP 120/84.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Oesophagus and stomach negative. The first part of the duodenum shows the presence of a redundant loop, of congenital origin, and no evidence of ulcer or stenosis."

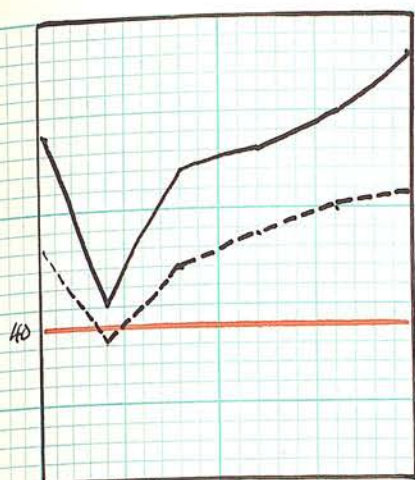
Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power and urea. (See graphs).

Urinary pH (See graphs).

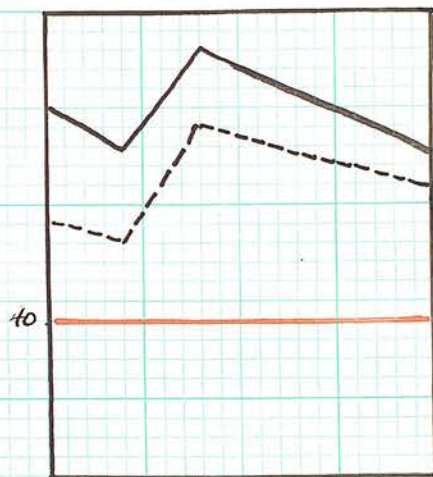
Notes: This man is suffering from pylorospasm, and probably duodenitis, in conjunction with a redundant duodenal loop. Etiologically the inflammation is probably associated with heavy smoking, but he does not confess to this, though it is borne out clinically and by observation. He improved rapidly under treatment, and has had no recurrence in the eight months since his discharge. Test meal shows severe hyperchlorhydria, which is considerably modified on the institution of treatment, and very profoundly modified after two weeks.

Conclusions: Clinically well: hyperchlorhydria
markedly modified by treatment. No alkalosis.
Controlled.

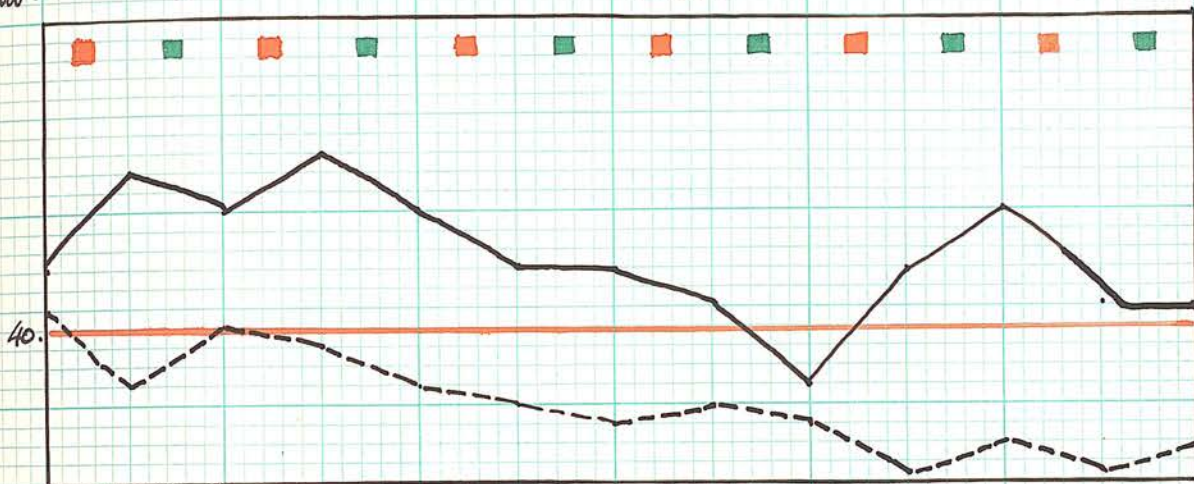
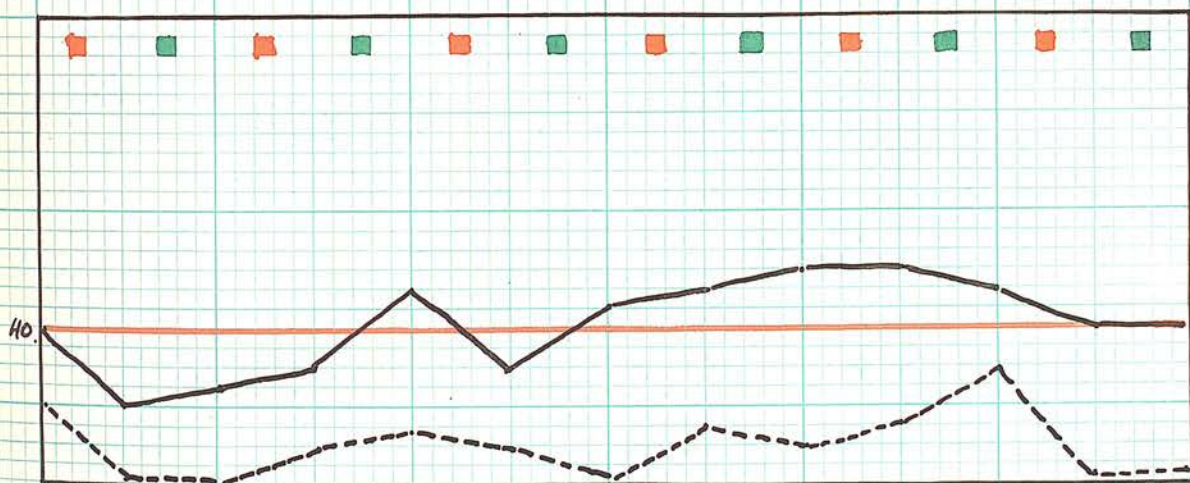


2-8-37, F-J 2024

STAFF	-	+	+	+	+	-
ONE	-	+	+	+	+	-
THREE	+	+	+	+	+	-
SIX	+	-	-	-	-	-



STARIN	+	+	+	+	+
BILE	-	-	-	-	-
OJOCOS	+	+	+	+	+
BLOOD	+	+	+	+	+

[illegible][illegible]

DAYS

3

6

9

12

15

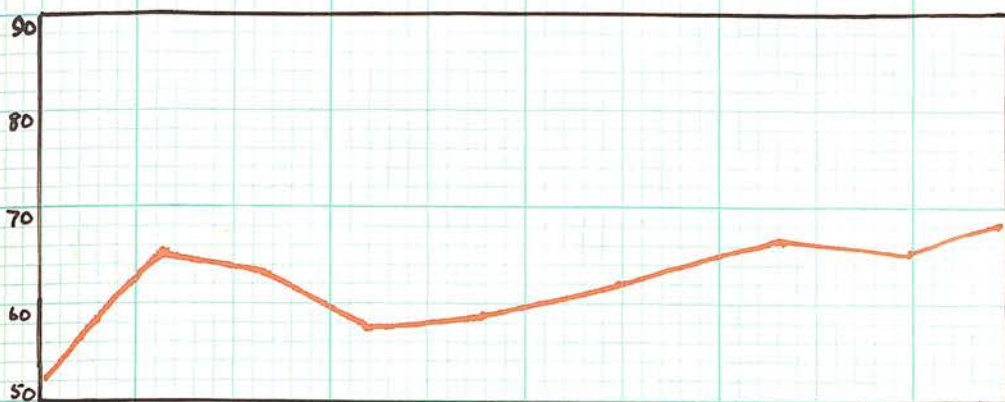
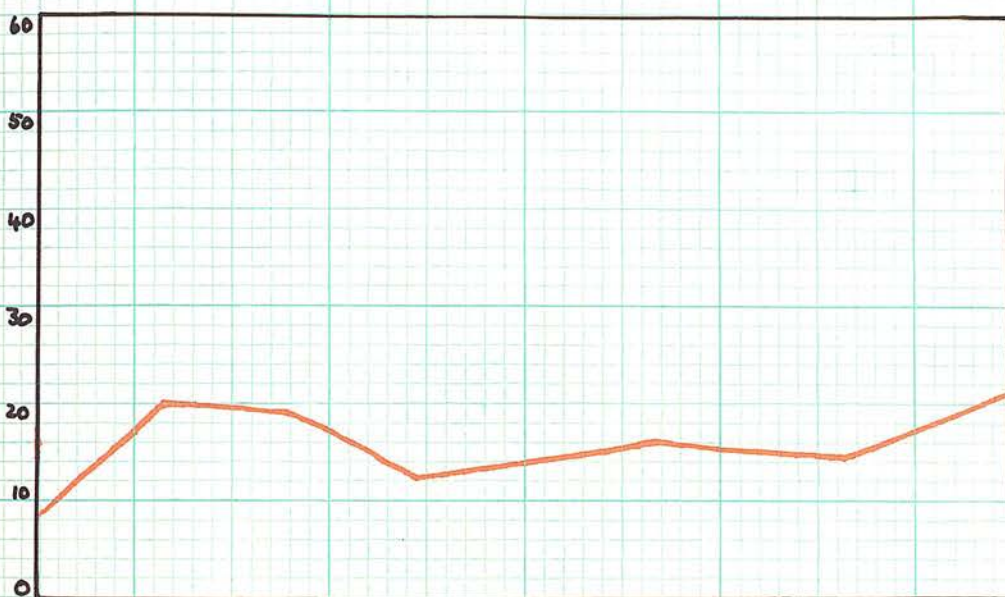
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21

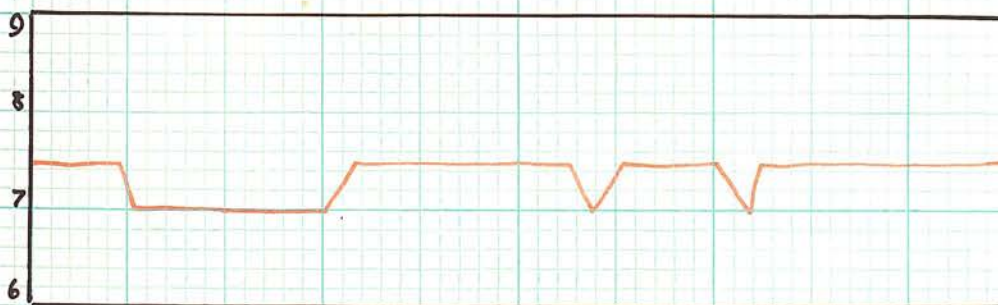
24

27

30

Blood CO₂ combining power (Vols. %)

Blood Urea (mgur. %)



Urinary pH.

Case (2) F.

James S. (aet. 34). Railway Porter.

Admitted: 15:1:38. Discharged: 10:2:38.

Complaint: "Pain in stomach" for twelve years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: He has had pain in the epigastrium intermittently for twelve years. It comes on 2-3 hours after a meal, is "gnawing" in character, and does not radiate. It is relieved by alkalis but not by food. He has periods free from pain and was once well for three years, but lately (three months) the pain has become much more persistent and severe, and he has had attacks of vomiting practically every day. The vomit was green and thick, but contained no blood. No heartburn or flatulence: he suffers a lot from acid eructations and is afraid to eat in case of starting the pain. He has lost weight. Bowels are costive.

Examination: He is a small, wiry, man, with a worried expression and rather "drawn" cheeks. No osseous deformity.

He is a non-smoker and teetotaler. He has a good house in the country.

Alimentary: Tongue dirty: throat clear: teeth - most have been removed, and the few remaining are carious.

Abdomen is thin and moves poorly on respiration.

There is some rigidity of the right upper rectus,

but no pain or tenderness on palpation. Splashing is easily elicited. The liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 130/94.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "No abnormality of stomach or oesophagus. The duodenal cap shows an ulcer deformity, there being a large ulcer crater at the apex on the lesser curvature side, indicating that the ulcer is active. No organic obstruction."

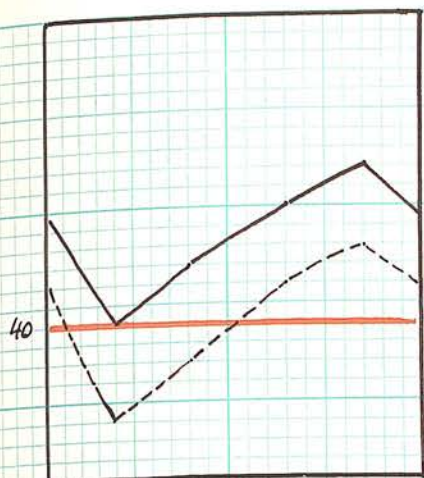
Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power and urea. (See graphs).

Urinary pH (See graphs).

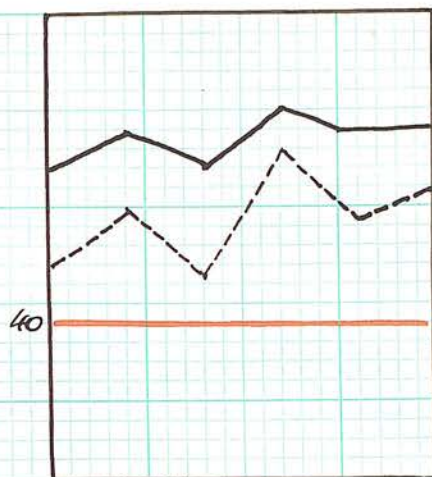
Notes: This man is suffering from the presence of a duodenal ulcer, confirmed radiologically. There was rapid clinical improvement on admission, and there has been no reflex since discharge eight months ago. Test meal shows a fairly severe degree of hyperchlorhydria, which was much modified by the institution of treatment: after two weeks, however, it was severe again.

Conclusions: Clinically well: hyperchlorhydria at first modified by treatment, then gradually rose again. No alkalosis. Not controlled.



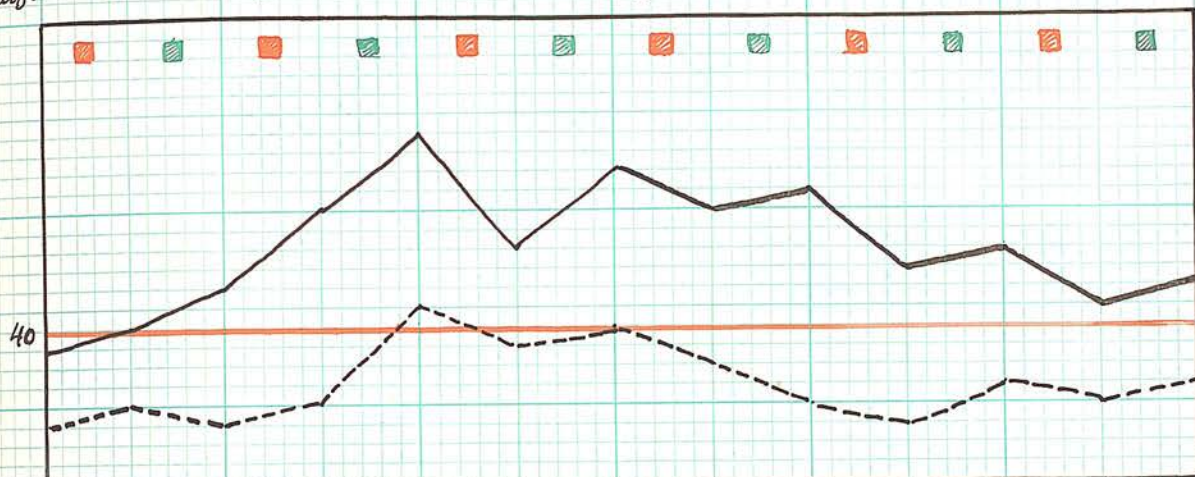
2.9.37. FV 204

SPRINT	+	+	+	+	+
BLUE	+	+	+	+	+
POCUS	+	+	+	+	+
BLOOD	+	+	+	+	+

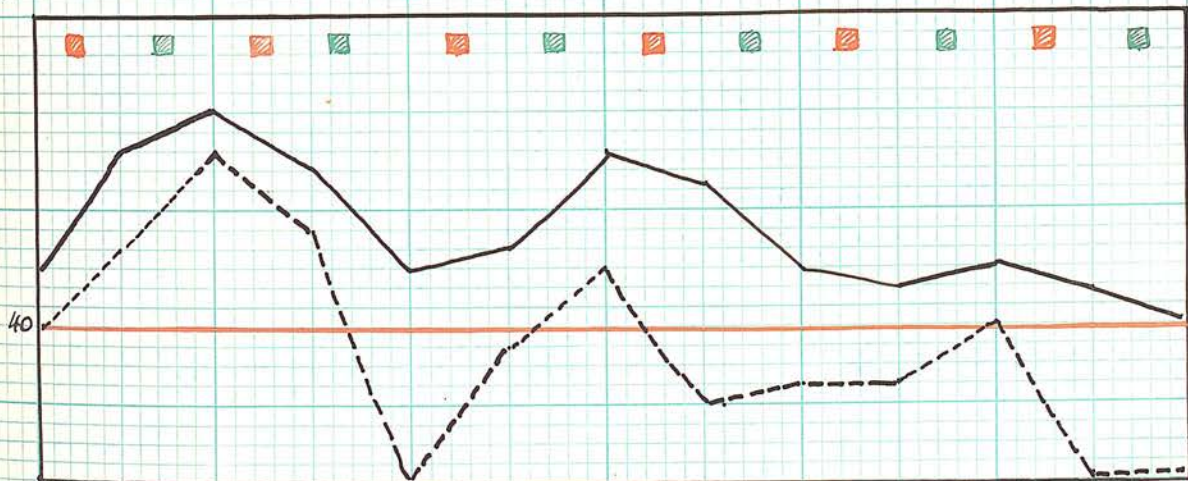


15.1.38 FJ30cc.

	15-1-58	17-1-58	18-1-58	19-1-58	20-1-58	21-1-58
STARCH	-	+	+	+	+	+
BILE	-	-	-	-	-	-
PROCES	+	+	-	-	+	+
BLOOD	-	-	-	-	-	-



17.1.38 FT 30CS.

[illegible]

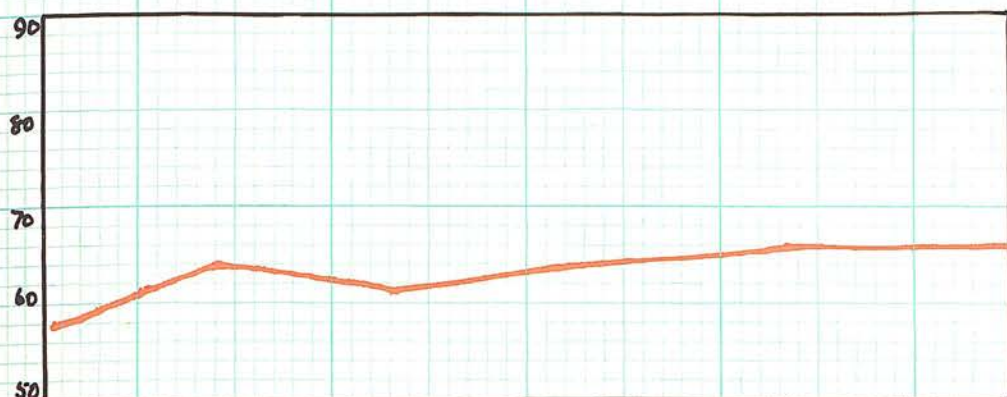
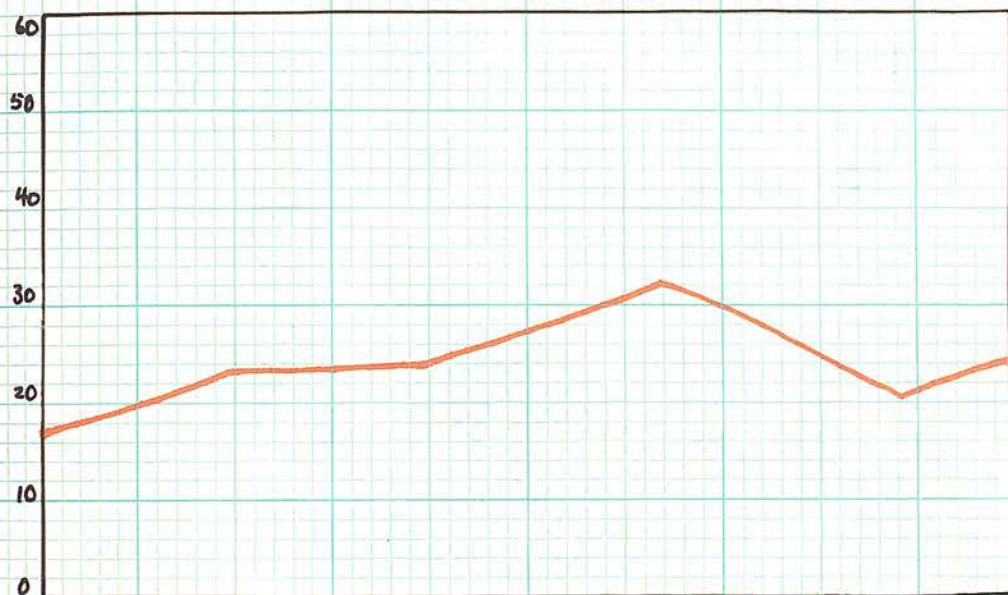
19.1.38. FJ 4044

STARCH +
BILE +
MUCUS +
BLOOD -

+ + + + + + + + + +

- - - - - - - - - -

DAYS. 3 6 9 12 15 18 21 24 27 30

Blood CO₂ combining power (Vols. %)

Blood Urea (mgm. %)



Urinary pH.

Case (2) G.

Roderick S. (aet.32). Locomotive Fireman.

Admitted: 17:1:38. Discharged: 17:2:38.

Complaint: Epigastric pain for three years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: For the past three years he has been troubled with a feeling of discomfort in the epigastrium, occasionally painful; recently it has been constantly painful. At times it has improved, but he has never been altogether free from it. It is a sharp pain, coming on 1-1½ hours after meals, and is relieved by food or alkali. It is felt a little to the right of the midline, below the ribs, and occasionally passes through to the back. It does not wake him at night. He has a fairly good appetite. About five weeks ago he had an attack of vomiting, but apart from this he has had no vomiting. He suffers considerably from flatulence, and passage of wind relieves the pain. Bowels are costive, and he has been losing weight recently.

Examination: He is a highly strung individual, fairly well built, and does not look ill.

He smokes 10 cigarettes per day, and drinks beer or spirits at the week-ends. He is married and has a good home.

Alimentary: Tongue dirty: teeth false: throat clear. Abdomen is well covered and moves well on respiration.

There is tenderness on palpation in the epigastric region, in the midline, and on removal of the examining hand, the pain persists. There is slight boarding of both recti in this region. No splashing was elicited. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 130/96.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "unsatisfactory film" (not repeated).

Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power and urea. (See graphs).

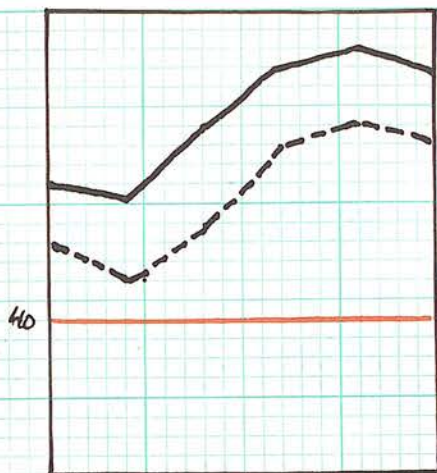
Urinary pH (See graphs).

Basal Metabolic +22.

Notes: This man is suffering from the presence of a duodenal ulcer, not definitely confirmed radiologically.

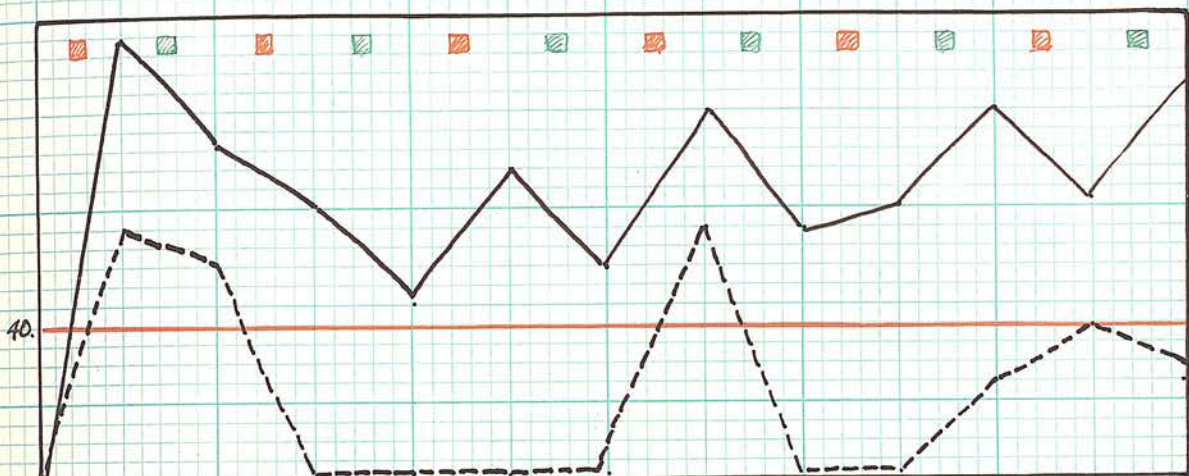
There was rapid improvement on admission, tenderness disappearing within six days, and no relapse within eight months of discharge. Test meal shows a severe degree of hyperchlorhydria, which is modified, though not to a great degree, by the institution of treatment: in two weeks, however, the hyperchlorhydria had again become very severe, though fluctuant.

Conclusions: Clinically well: hyperchlorhydria modified at first by treatment, uninfluenced after two weeks. No alkalosis. Not controlled.



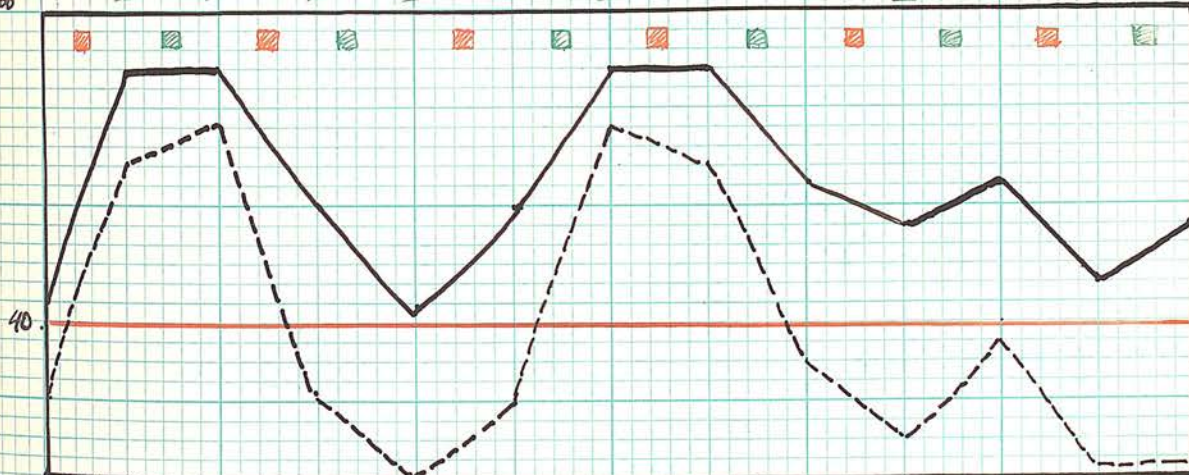
18.1.38 FT 40cc

STARCH
BILE
MUCUS
BLOOD.



19.1.38 FT 30cc

STARCH
BILE
MUCUS
BLOOD

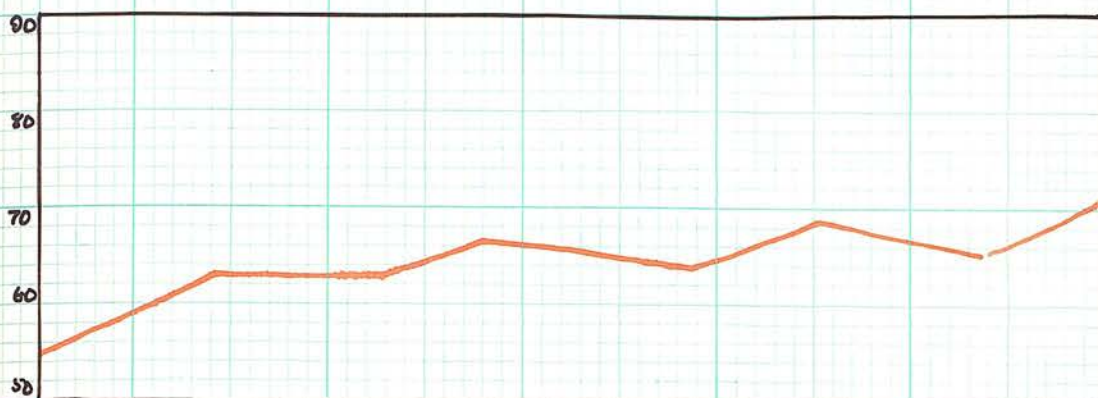
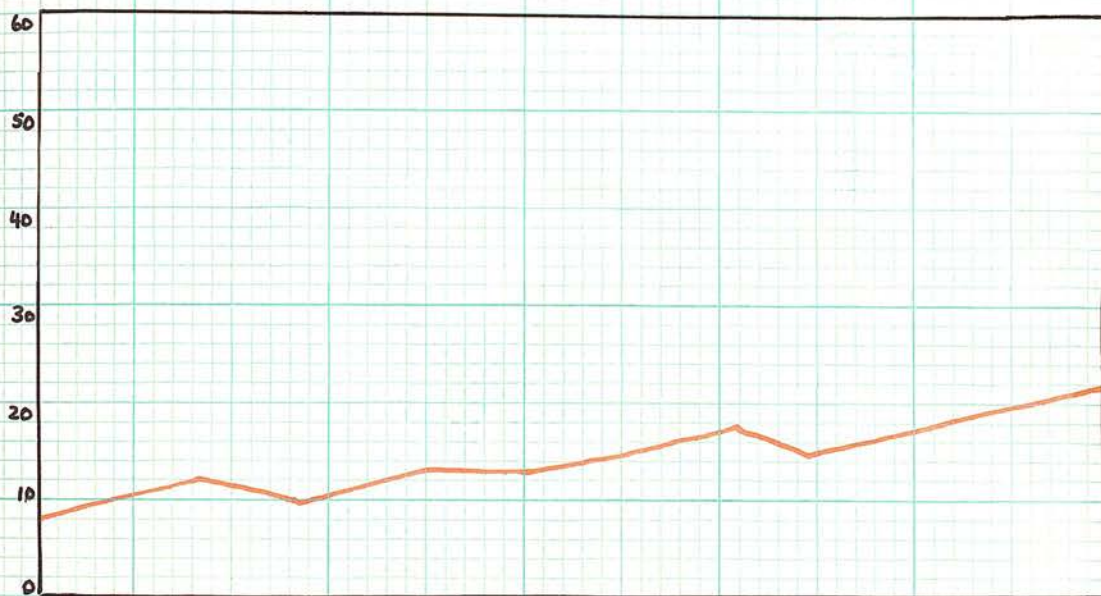


19.1.38 FT 40cc

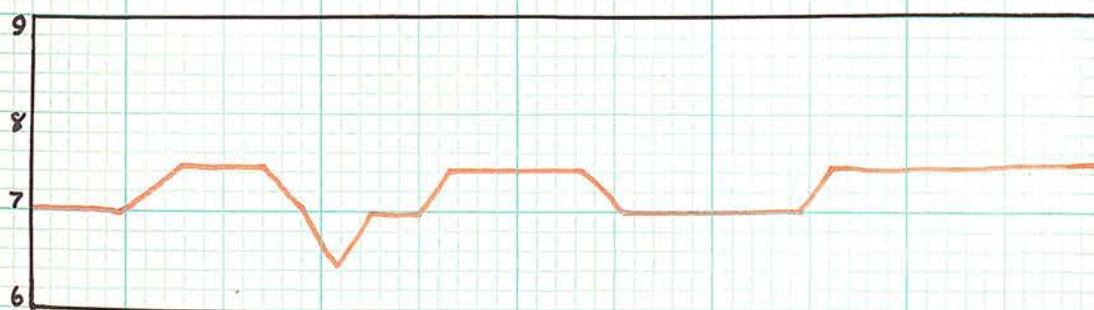
STARCH
BILE
MUCUS
BLOOD



DAYS. 0 3 6 9 12 15 18 21 24 27 30

Blood CO₂ combining power (Vols. %)

Blood urea (Mgm. %)



Urinary pH

Case (2) H.

William C. (aet. 46). Cabinet Maker.

Admitted: 17:5:38. Discharged: 17:6:38.

Complaint: Severe epigastric pain for three months,

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: Until three months ago, he was perfectly well.

One day he vomited a small quantity of blood, bright red, and for a day or two afterwards the stools were black. He became pale and dizzy. Since then he has been very pale and has lost a lot of weight, and epigastric pain has developed and become very severe. He has occasional attacks of vomiting of green-stained infested food. The pain is now constant and not relieved by food or alkali, though previously it came on about $1\frac{1}{2}$ -2 hours after food and was relieved by food or alkali. There is a good deal of heart-burn and flatulence. He is now never free from pain, and wakes in the night, troubled by it.

Examination: He is a pale, undersized man, with a marked upper dorsal kyphosis and scoliosis with convexity to the right which dates from childhood. He has lupus erythematosus of the face.

He smokes 10-15 cigarettes per day, and is a teetotaller. He is married, and in comfortable circumstances.

Alimentary: Tongue clean: teeth false: throat clear.

Abdomen is thin, but moves well on respiration.



There is slight tenderness in the midline in the epigastrium, but no rigidity. Splashing is not elicited. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 140/100.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

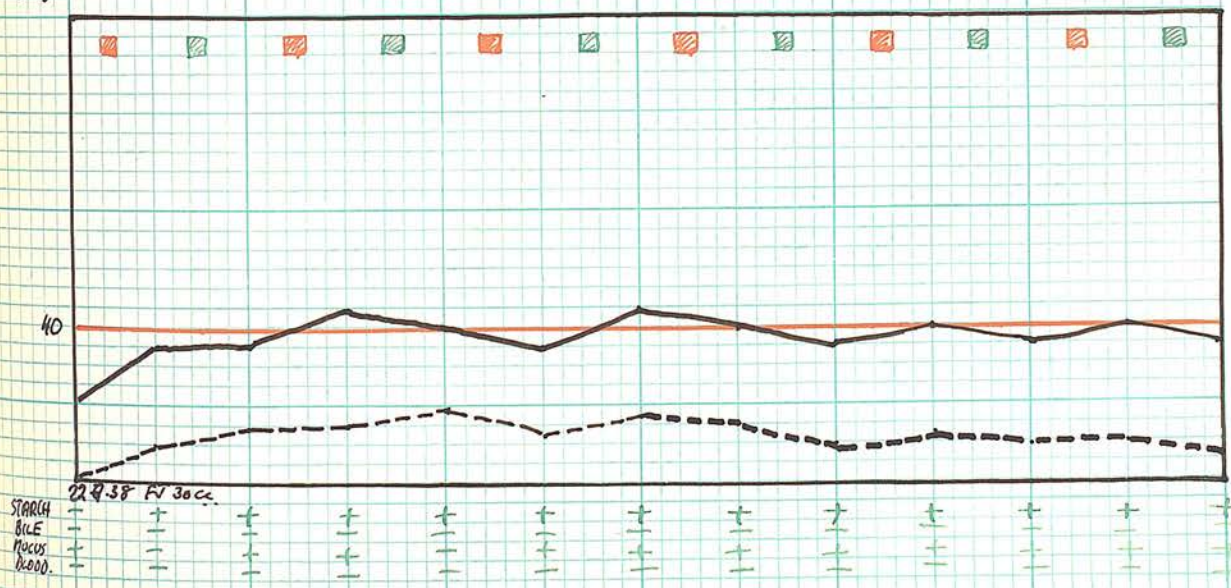
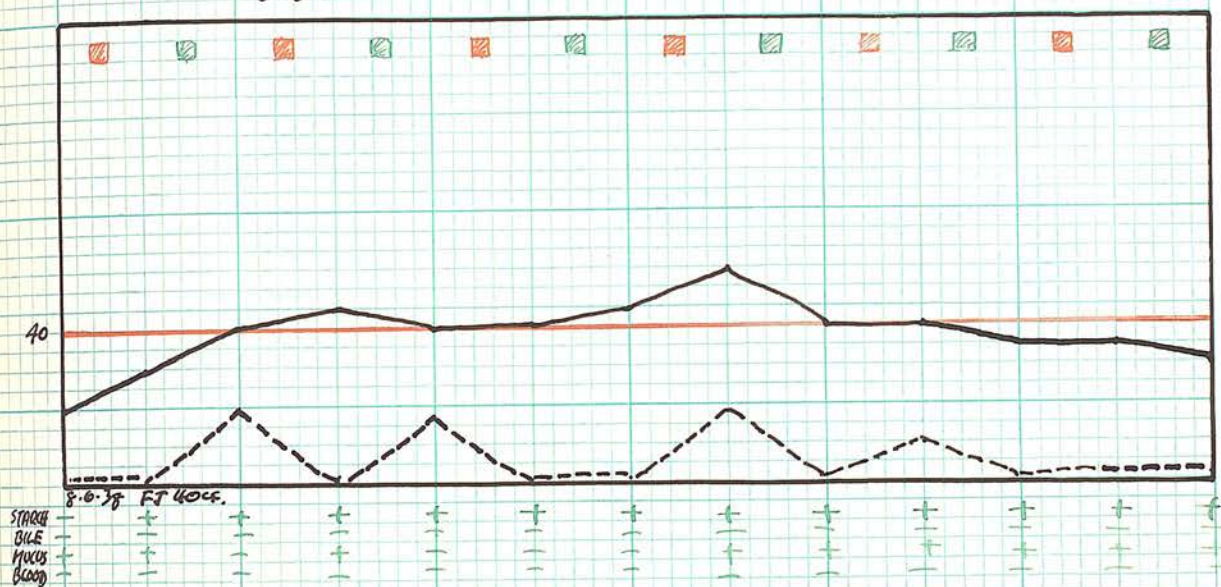
Wassermann test negative.

Benzidine reaction of stools negative (done every second day).

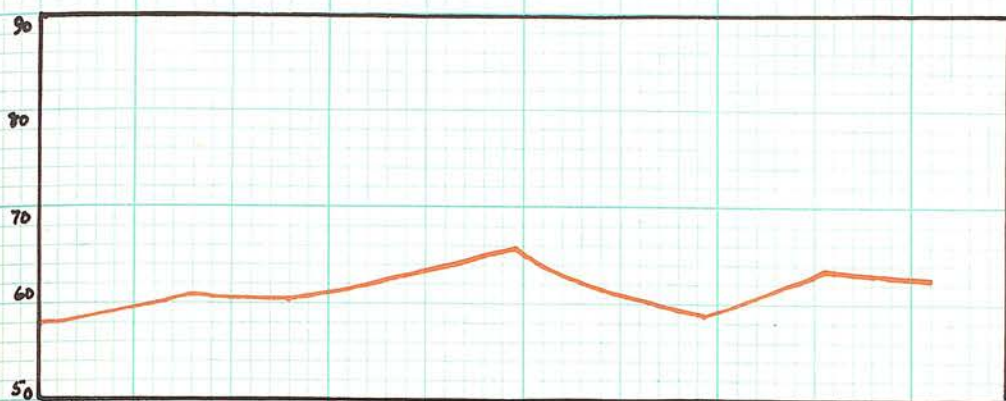
X-Ray shows "Stomach held high below costal margin and cannot be palpated. Body of stomach and peristalsis complete to pylorus. Duodenal cap directed backwards and can only be seen in lateral view - appears normal. No definite signs of ulceration. Urine negative for casts, albumen etc. (done daily). Blood CO₂ combining power and urea (See graphs). Urinary pH (See graphs).

Notes: This man is suffering from the presence of a duodenal ulcer, not confirmed radiologically. Rapid improvement ensued from the day of admission, and there was no relapse three months after discharge. Test meal shows a moderate hyperchlorhydria, associated with a "climbing curve." This is markedly modified by the institution of treatment, and the improvement maintained after two weeks.

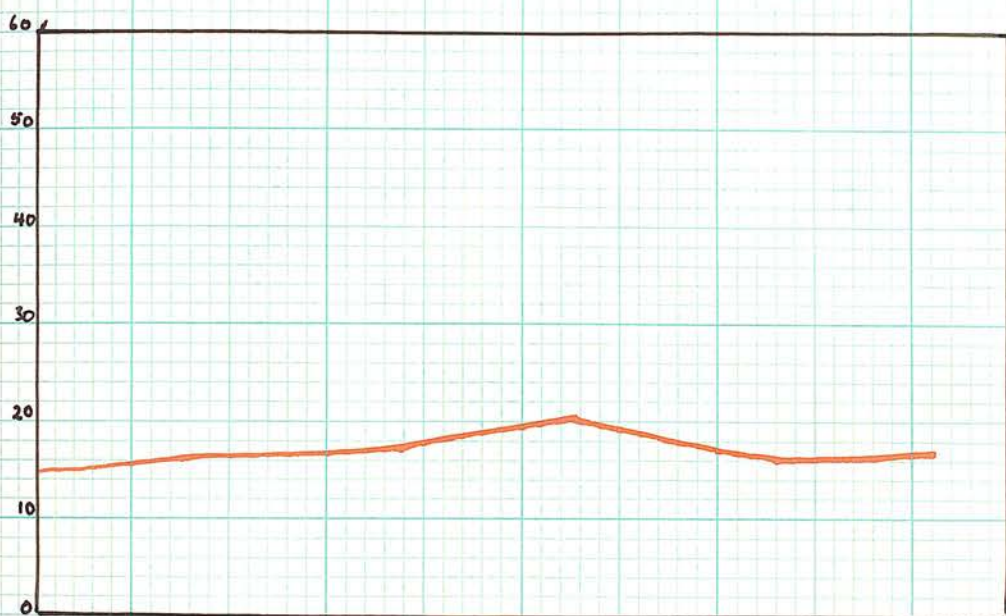
Conclusions: Clinically well: hyperchlorhydria markedly modified by treatment. No alkalosis. Controlled.



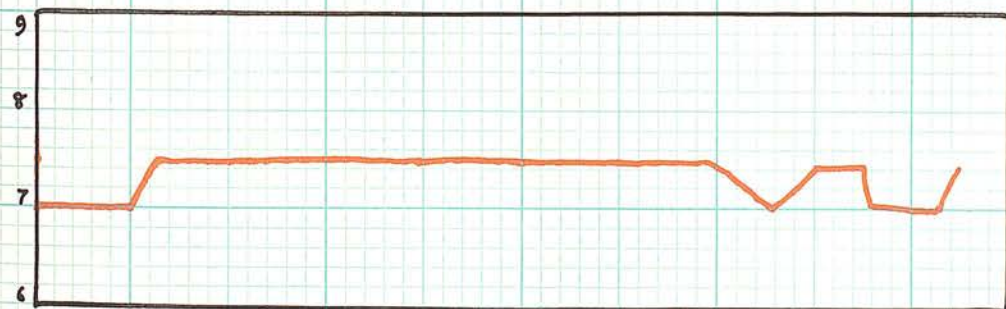
DAYS 0 3 6 9 12 15 18 21 24 27 30



Blood CO₂ combining power (Vol-%)



Blood urea (mgm-%)



Urinary pH

Case (2) I.

Thomas McK. (aet. 38). Spice Miller.

Admitted: 2:5:38. Discharged: 29:5:38.

Complaint: Epigastric pain intermittenly for seventeen years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: Seventeen years ago he began to suffer from indigestion. At that time he was having meals at irregular intervals and travelling long distances by car. Epigastric pain unrelated to food was the main symptom and there were frequent remissions. Ten years ago his appendix was removed and he was better for a few weeks. Since then he has got gradually worse. Pain appears about three hours after taking food, and is very severe during the night. It is not relieved by the taking of food or alkali. There is no vomiting, and flatulence is not marked. Acid eructations are frequent, and excessive salivation is troublesome. Bowels are constipated. He has lost about one stone in weight, during the last year.

Examination: He is a poorly built sallow individual. There is no obvious osseous deformity.

He smokes a lot (over 20 cigarettes per day), and occasionally drinks beer.

Alimentary: Tongue heavily coated: teeth false: throat clear. Abdomen is thin, and moves poorly on respiration.

It is held resistant to palpation, especially of the epigastric region. There is considerable tenderness in the epigastrium to the left of the midline, and pain on withdrawal of the examining hand. The left upper rectus shows a slight degree of rigidity. Splashing is elicited fairly easily. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 125/70.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach J-shaped; good tone, peristalsis and motility. No abnormality seen. Duodenal cap considerably deformed but no definite niche seen."

X-Ray of gall bladder "No abnormality in straight film. Sixteen hours after dye, gall bladder well outlined. After fatty meal, shadow smaller.

Functioning gall bladder; no evidence of calculi."

Urine negative for casts, albumen etc. (done daily).

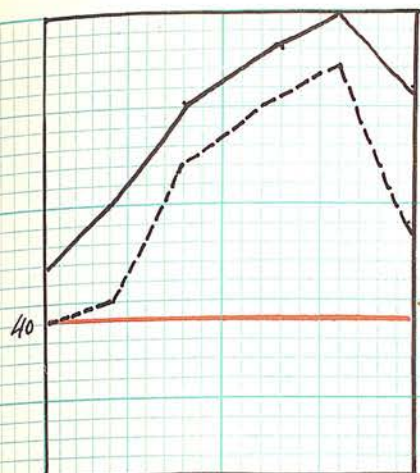
Blood CO₂ combining power and urea (See graphs).

Urinary pH (See graphs).

Notes: This man is suffering from the presence of a duodenal ulcer, not definitely confirmed radiologically. (He pointed out himself that his work - Spice

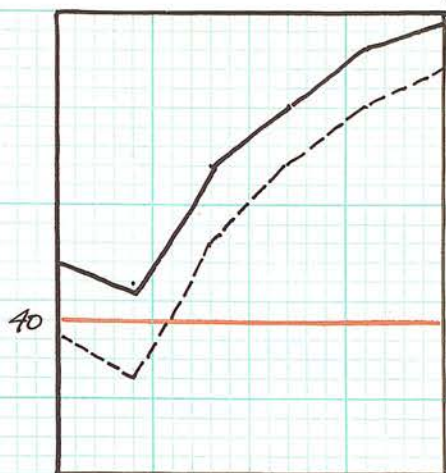
Miller - involves the involuntary partaking of considerable quantities of McLean's Power, aloes, etc!) He improved rapidly in hospital, and has had no relapse in the three months since discharge. Test meal shows a fairly severe degree of hyperchlorhydria associated with a "climbing" curve. This is modified to a certain extent, after the institution of treatment, and no further modification is attained after two weeks.

Conclusions: Clinically well: hyperchlorhydria modified to a certain degree by treatment. No alkalosis. Partially controlled.



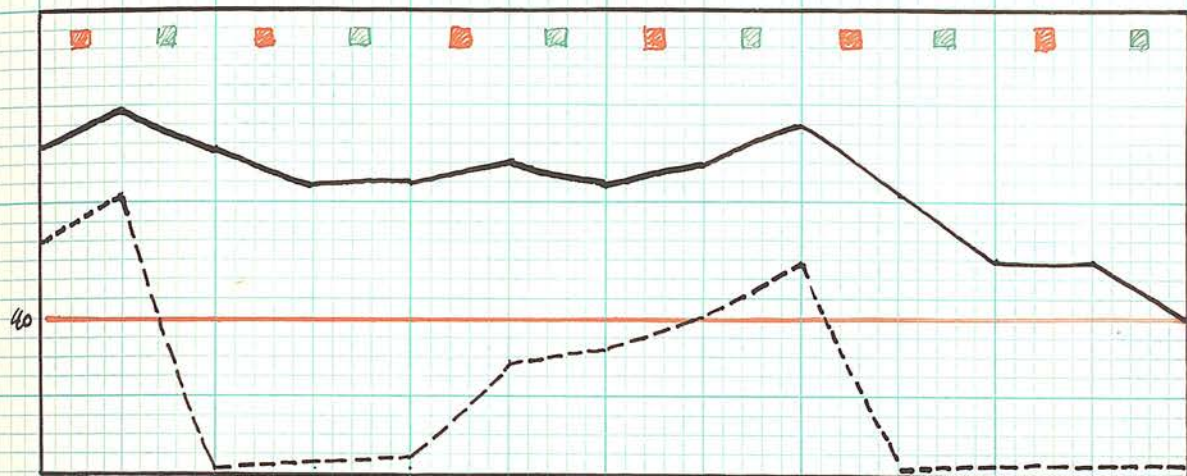
2.2.38 FJ 60 CS.

STARCH	-	+	+	+	+	+
BILE	-	-	-	-	-	-
MUCUS	+	+	+	+	+	+
BLOOD	-	-	-	-	-	-

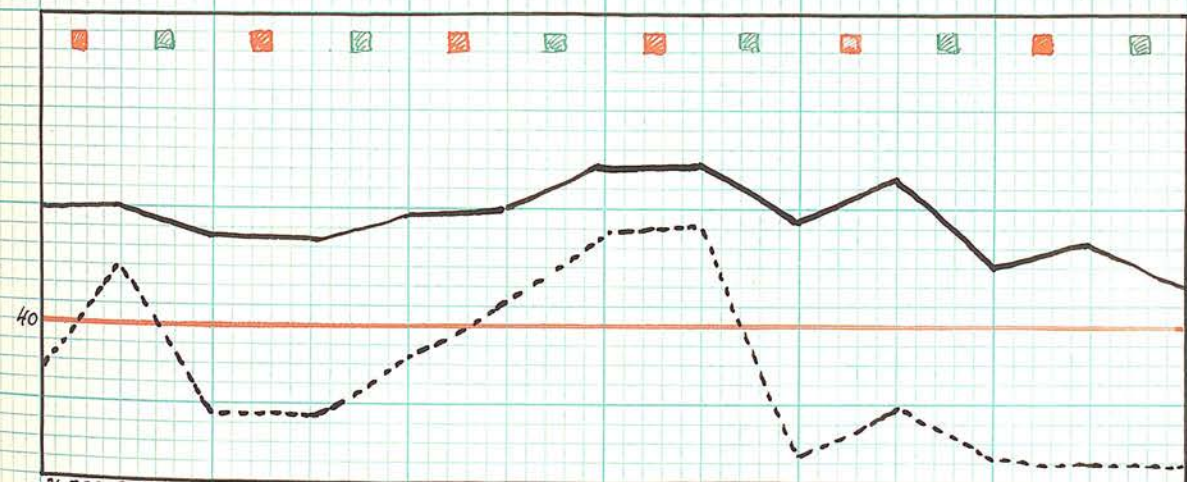


5.5.38 ET 60 cc

STARCH	-	+	+	+	+
BILE	-	-	-	-	-
TRIGL	+	+	+	+	+
BLOOD	-	-	-	-	-



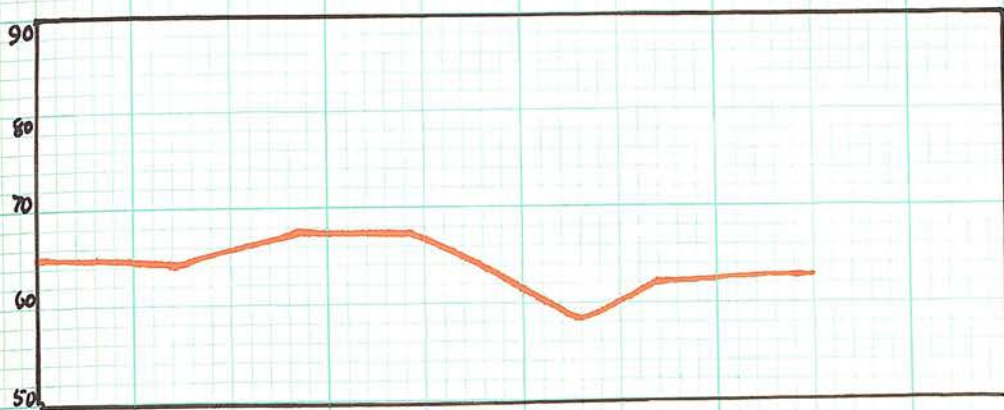
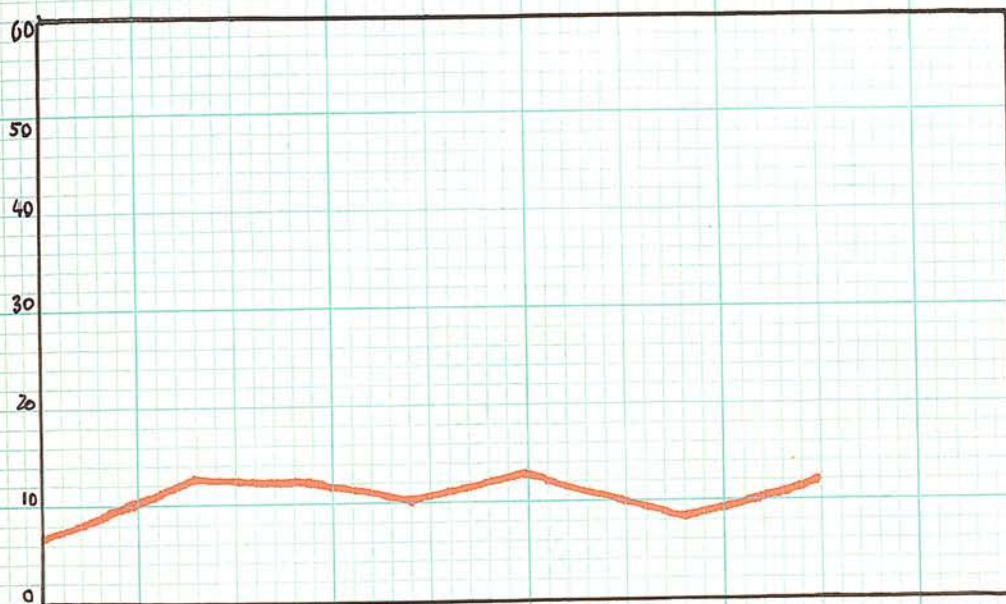
7.5.38 FJ 46cc

[illegible]

21.5.38 FJ 60°C.

[illegible]

DAYS 0 3 6 9 12 15 18 21 24 27 30

Blood CO₂ combining power (Vol %)

Blood urea (mgm. %)



Urinary pH.

Case (2) J.

Mary B. (aet 32). Housewife.

Admitted: 9:4:38. Discharged: 8:5:38.

Complaint: Epigastric pain for one year.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: For the past year she has suffered from epigastric pain, which comes on 1-1½ hours after food, and is usually accompanied by a feeling of nausea. It is not relieved by the taking of alkali or food, but it is relieved by vomiting. It is sharp in character and sometimes felt in the back.

She suffers also from flatulence and acid eructations and heartburn. The pain is getting more severe, and staying much longer than it did at first. She has lost some weight. Bowels are regular; menstrual periods are regular.

Examination: She is a well built woman, and does not show any signs of loss of weight. There is no obvious osseous abnormality.

Alimentary: Teeth false: tongue dirty: throat clear. Abdomen is well covered and moves freely. There is some tenderness below the right costal margin about 1" from the midline, but no muscle rigidity. Splashing is elicited easily. Liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 110/84.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine test of stools negative (done every second day).

X-Ray shows "Atonic stomach containing a fair amount of residual fluid. Peristalsis complete to pylorus, but not very effective for a time - pylorospasm.

Duodenal cap deformed and shows presence of niche - duodenal ulcer."

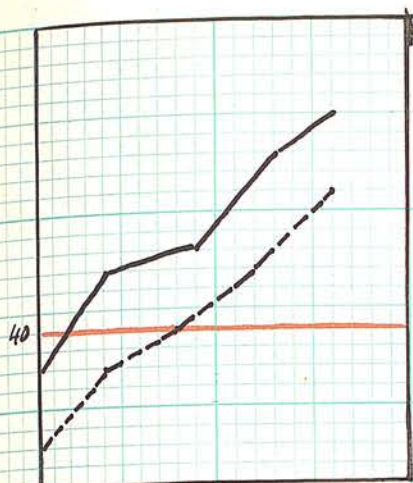
Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power and urea (See graphs).

Urinary pH (See graphs).

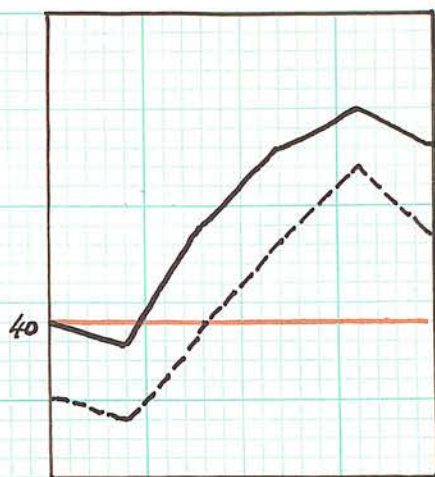
Notes: This woman is suffering from the presence of a duodenal ulcer, confirmed radiologically. She improved rapidly in hospital, and tenderness disappeared in five days. There has been no relapse since discharge three months ago. Test meal shows severe hyperchlorhydria associated with a "climbing" curve. This is modified a little on the institution of treatment, and a little more after two weeks, but is still very severe.

Conclusions: Clinically well: hyperchlorhydria severe and little modified by treatment. No alkalosis. Not controlled.



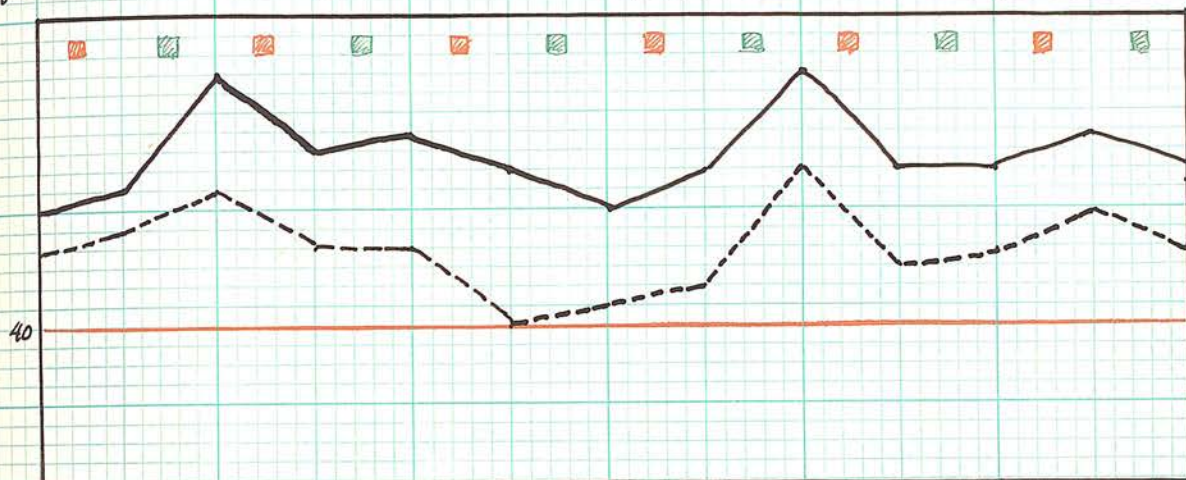
2.2.38 FT30CC.

STARCH	+	+	+	+
BILE	+	+	+	+
MUCUS	+	+	+	+
BLOOD	+	+	+	+

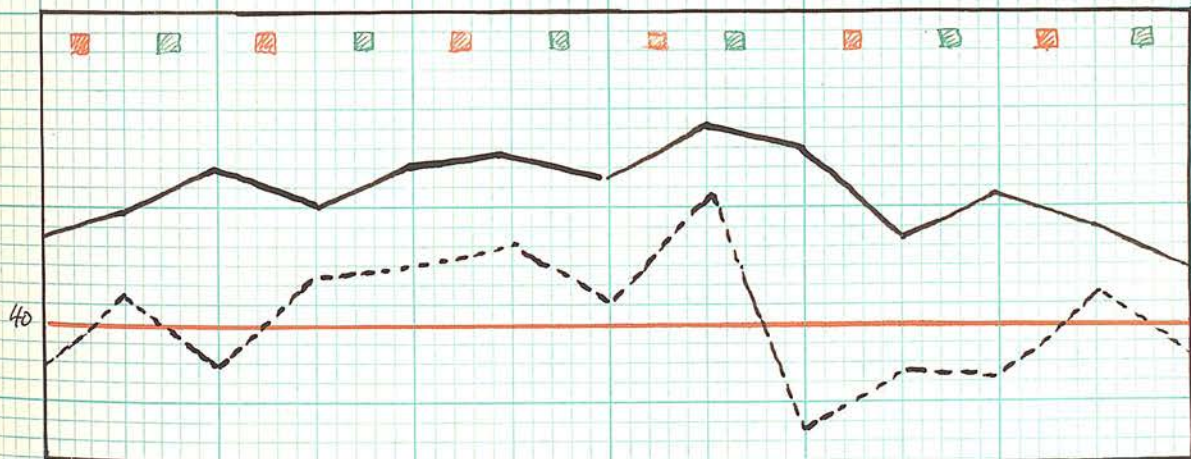


5.438 FJ 40 cc.

STARCH	+	+	+	+	-
BILE	-	-	-	-	-
MUCUS	+	+	+	+	+
BLOOD	-	-	-	-	-



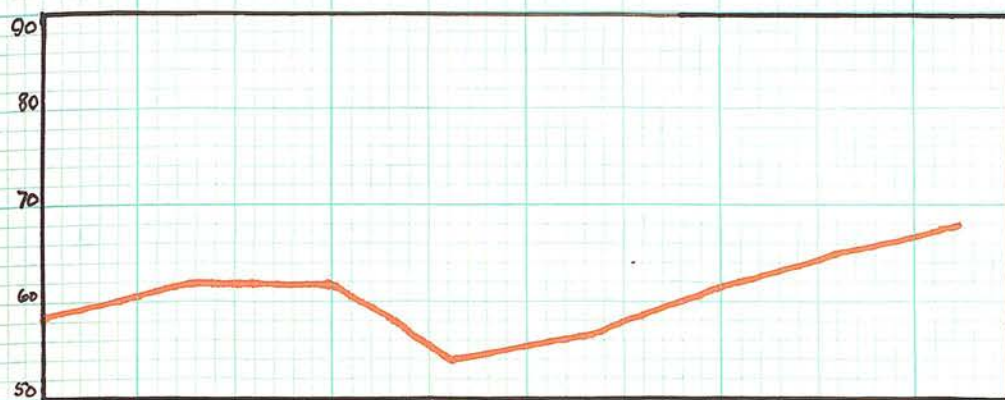
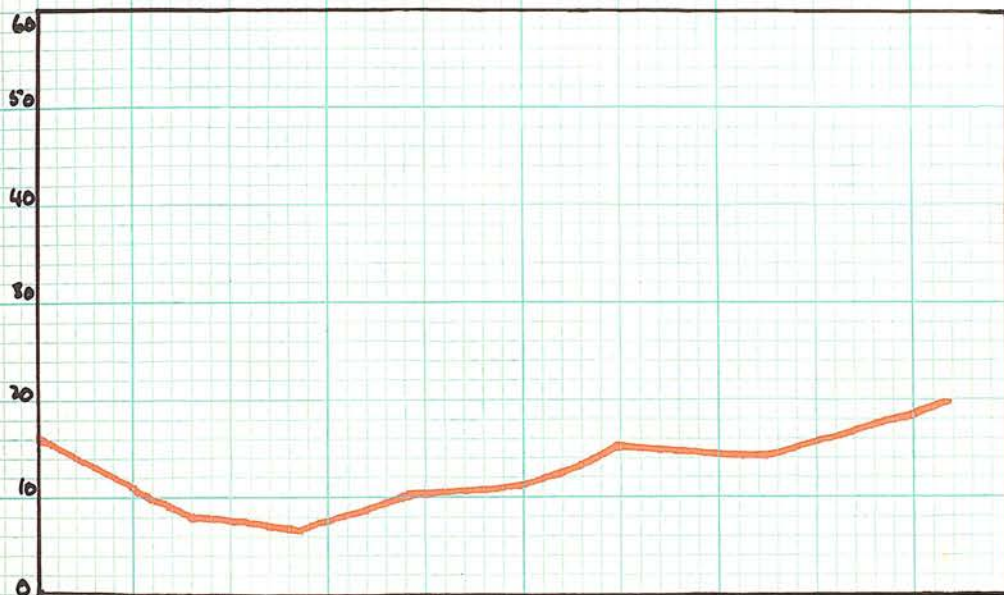
6.4.38 FJ 30cc.

[illegible]

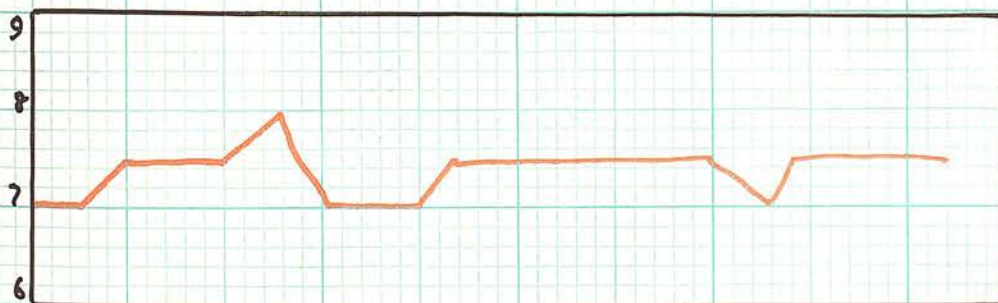
22.4.38 FJ 20cc.

[illegible]

DAYS 0 3 6 9 12 15 18 21 24 27 30

Blood CO₂ combining power (Vol-%)

Blood urea (mgm-%)



Urinary pH.

Case (2) K.

William V. (aet. 29). Railway Worker.

Admitted: 27:2:38. Discharged: 10:4:38.

Complaint: Epigastric pain for nine months.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-alkali.

History: This man has suffered from epigastric pain for nine months. It is gnawing in character, comes on 2-3 hours after meals, and is relieved by taking food or alkali. There is no vomiting, though he sometimes feels sick. The pain is getting worse recently, and he is losing weight. He occasionally feels it in his back, and has felt it travel up to his right shoulder. He is very constipated. He has had no heartburn, but a good deal of flatulence, which seems to relieve the pain.

Examination: He is well built and does not look as though he had lost weight, but he has an anxious expression. No obvious osseous abnormality.

Alimentary: Tongue clean: teeth good: throat clear. Abdomen is well covered, and moves freely on respiration. There is no tenderness or rigidity on palpation. Splashing is not elicited. Liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 110/90.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine test of stools negative (done every second day).

X-Ray shows "J-shaped stomach with hyperactive peristalsis. Motility fair, good tone, no abnormality of wall. Duodenal cap persistently deformed and niche seen.

Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power and urea (See graphs).

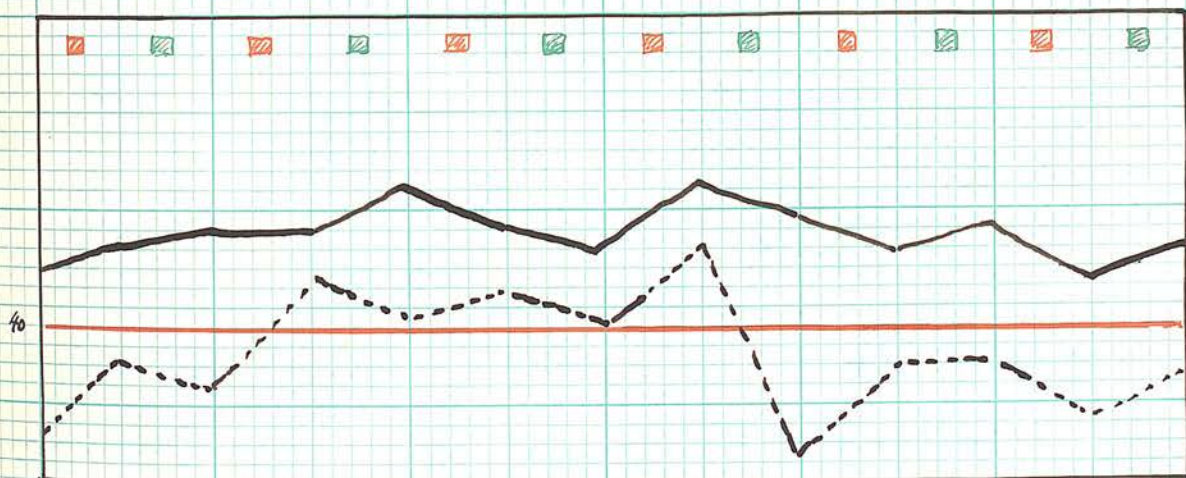
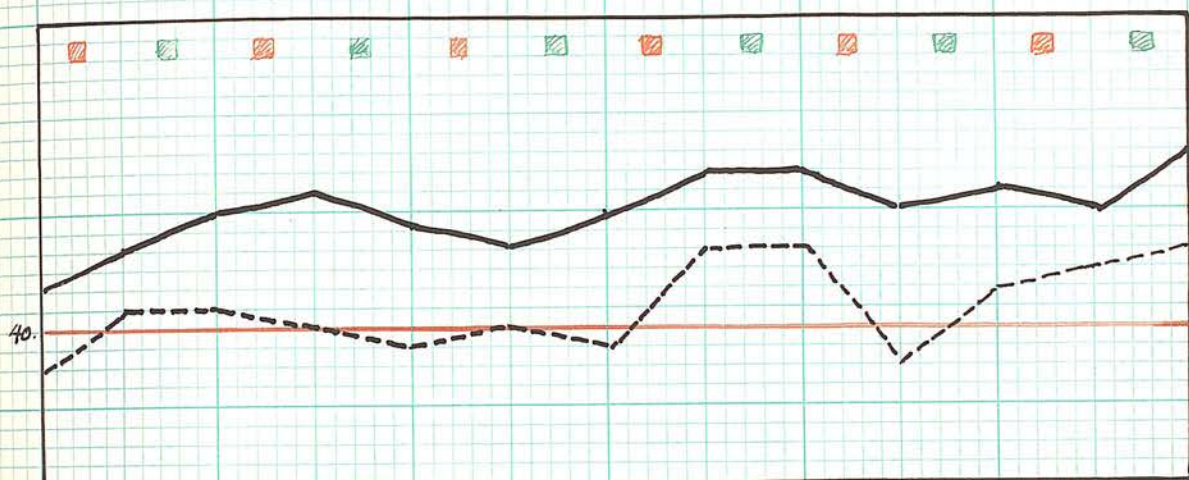
Urinary pH (See graphs).

Notes: This man is suffering from the presence of a duodenal ulcer, confirmed radiologically. He improved rapidly in hospital, and has had no recurrence of symptoms since discharge five months ago.

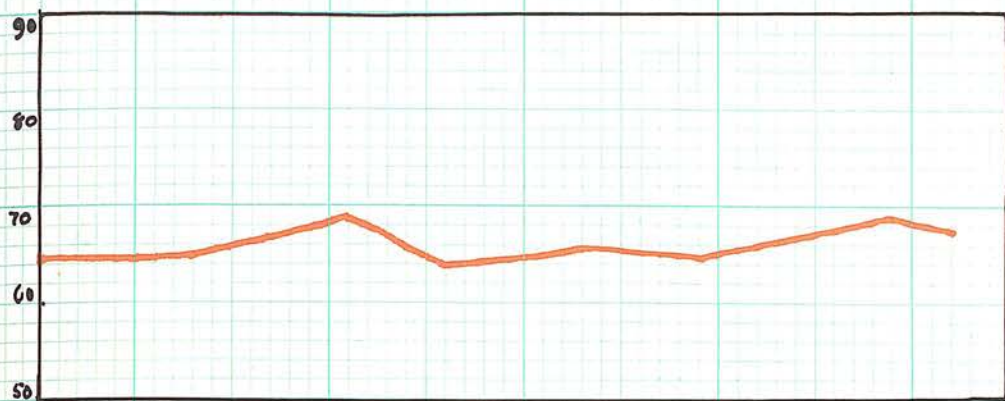
Test meal shows hyperchlorhydria associated with a "climbing" curve. This is slightly modified by treatment, but is still consistently high after two weeks.

Conclusions: Clinically well: hyperchlorhydria little influenced by treatment. No alkalosis. Not controlled.

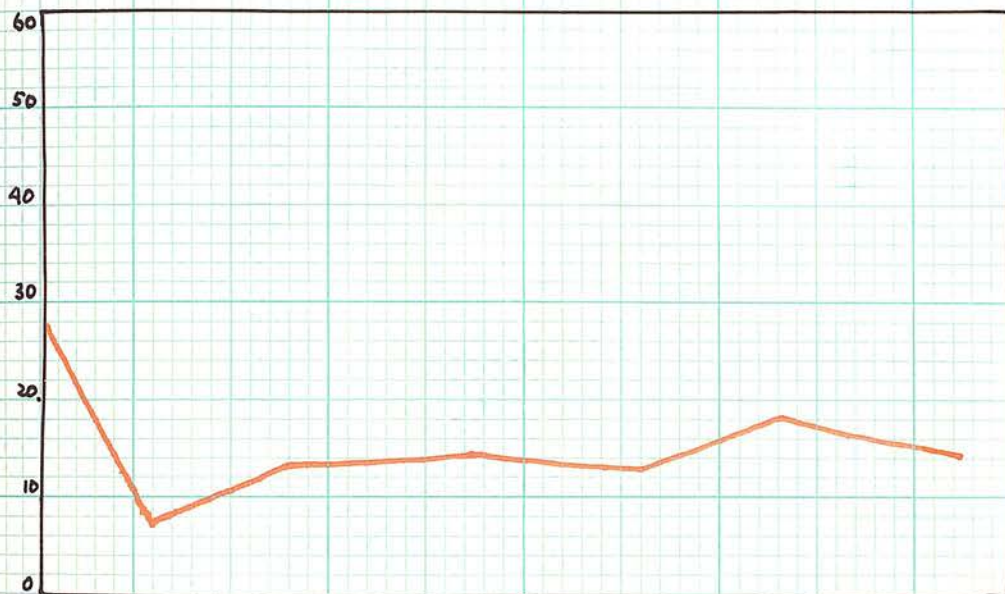
2. K.



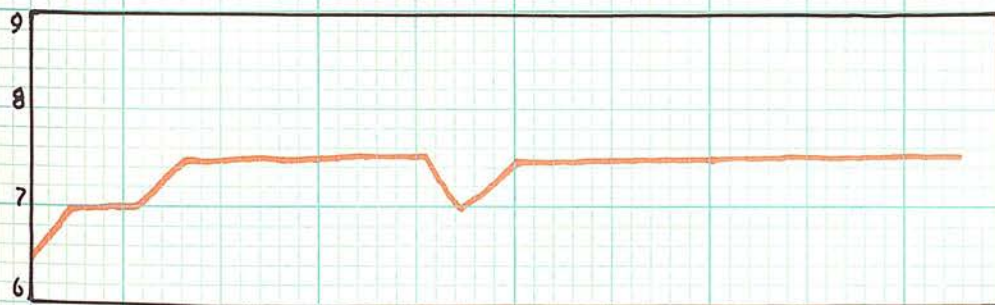
DAYS 0 3 6 9 12 15 18 21 24 27 30



Blood CO₂ combining power (vol%.)



Blood urea (mgm. %)



Urinary pH

Case (3) A.

Kenneth C. (aet. 35). Oxyacetylene Welder.

Admitted: 7:6:38. Discharged: 29:6:38.Complaint: Epigastric pain for 19 years.Diagnosis: Duodenal ulcer.Treatment: Sippy-magsorbent.

History: Since the age of sixteen this man has been troubled by epigastric pain, coming on two hours after food, and by flatulence and occasional attacks of vomiting, which sometimes relieved the pain. From time to time temporary relief has been afforded by alkali powders. He was treated for haematemesis seven years ago, and for perforated gastric ulcer three years ago. After these catastrophes he had relief for a few months, but relapsed again. He was relatively well until three months ago, when he had recurrence of the pain, accompanied by vomiting of small quantities of very sour material several times a day. He has got gradually worse. Bowels are constipated: he has lost weight.

Examination: He is well built, but pale and anxious. No obvious osseous deformity.

He smokes 15 cigarettes daily, and is unmarried and a teetotaller.

Alimentary: A few lower teeth are still present and carious. Tongue clean: throat clear. Abdomen moves well on respiration. There is no rigidity apparent,

though pain is still present. Liver and spleen are not enlarged. Splashing is not elicited.

Cardiovascular: Nil to note. BP 120/70.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach and oesophagus negative. Duodenal cap is markedly deformed, and right base particularly is difficult to clear, but no definite ulcer crater can be made out. Deformity the result of ulceration.

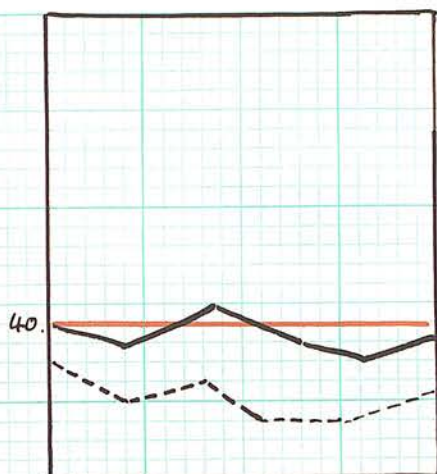
Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power 68 vols. %

Urinary pH varied between 6.5 and 7.5.

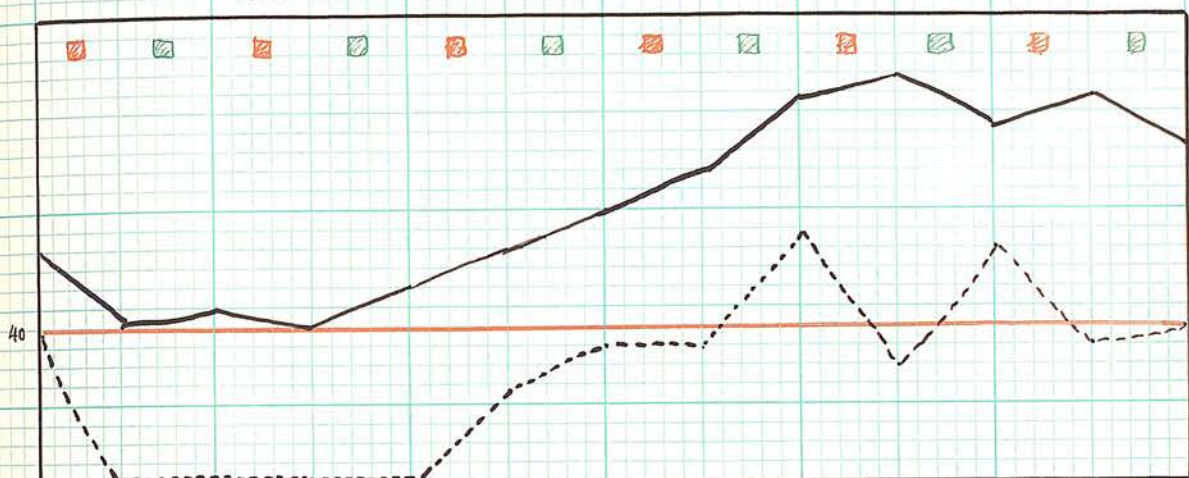
Notes: This man is suffering from the presence of a duodenal ulcer, confirmed radiologically. He improved rapidly in hospital, and has had no recurrence of symptoms since discharge three months ago. Test meal shows acidity to be within normal limits, but the long test meal shows that there is a tendency for the acidity to rise late in the day, and this is not altered after two weeks treatment.

Conclusions: Clinically well: acidity within normal limits, and well controlled.

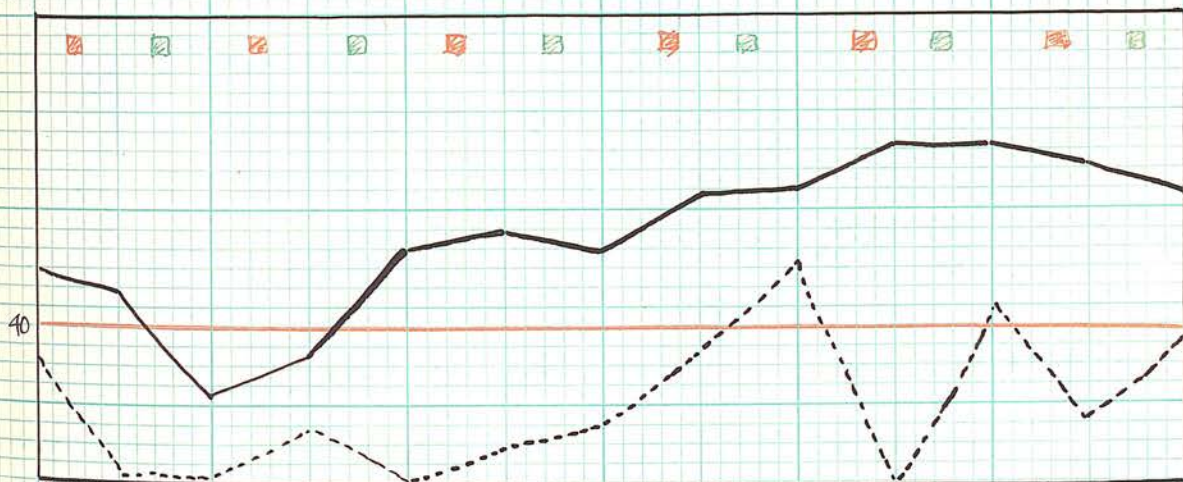


9-6-38 FJ 10CC.

STARCH	-	+	+	+	+
BILE	-	-	-	-	-
MUCUS	+	+	+	+	+
BLOOD	-	-	-	-	-



10.6.38 FJ 10cc.

[illegible]

26.6.38 FJ204

[illegible]

Case (3) B.

John MacL. (aet. 19). Apprentice Draughtsman.

Admitted: 24:5:38. Discharged: 18:6:38.

Complaint: Passage of black stools for one week.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-Magsorbent.

History: This youth was treated a year ago for haematemesis, the result being satisfactory. He remained well save, with practically no digestive upset, until about ten days ago, when he suddenly felt dizzy in the street. He fainted, and had an incontinent black motion. He was removed home and put to bed and transferred to hospital a few days later. He has had no pain or discomfort of any kind, and the motions are now normal in colour. He has been taking no medicines.

Examination: He is pale, but well built and does not look ill.

He is a teetotaller and a non-smoker.

Alimentary: Teeth - one or two are decayed: tongue clean: throat clear. Abdomen well covered and moves freely on respiration. There is no pain, rigidity or tenderness. No splashing is elicited. Liver and spleen are not enlarged.

Cardiovascular: Nil to note. BP 100/60.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann test negative.

Benzidine reaction of stools positive for three days, negative thereafter.

X-Ray "Stomach and oesophagus negative. Duodenal cap deformed and ulcer crater seen on greater curvature side."

Urine negative for casts, albumen etc. (done daily).

Blood CO₂ combining power 59.5 vols.

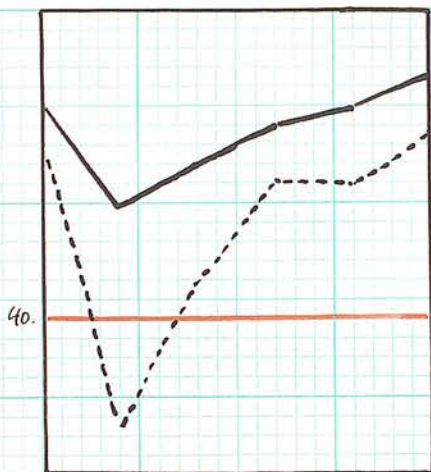
Urinary pH varied from 6.5 to 7.5.

Note: From 9:6:38 till 13:6:38 he continued on Sippy regime with the substitution of alkali for magsorbent. A test meal was then done and the alkali stopped and magsorbent recommenced.

Notes: This boy is suffering from duodenal ulceration, confirmed radiologically. He has few symptoms, but has probably been bleeding from the ulcer. He felt well in hospital and has had no recurrence since discharge, three months ago.

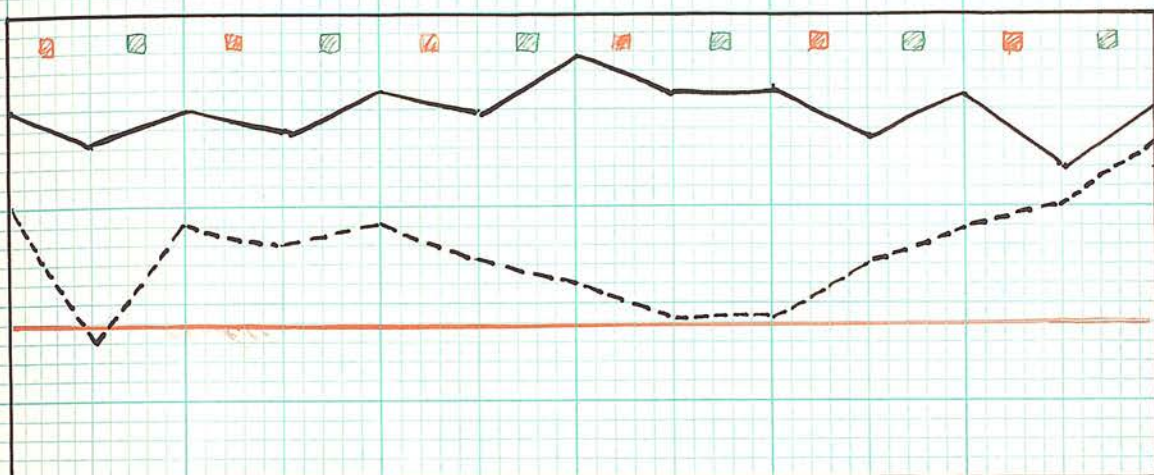
Test meal shows hyperchlorhydria, and this is completely uninfluenced by Sippy treatment, using either Magsorbent or Alkali, and shows no alteration after three weeks.

Conclusions: Clinically well: severe hyperchlorhydria, unaffected by treatment or drugs. Not controlled.



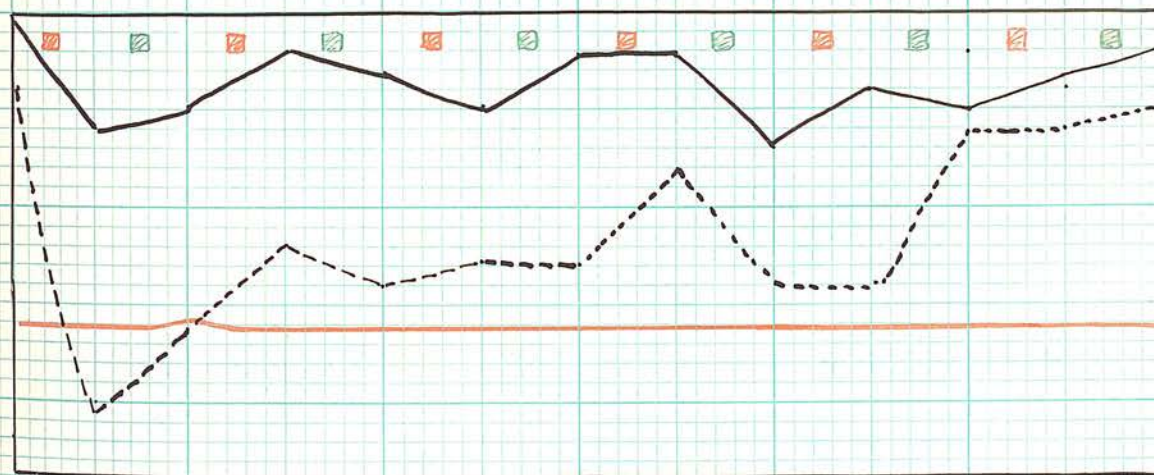
24.5.38 FT 20cc.

STARCH
BILE
MUCUS
BLOOD



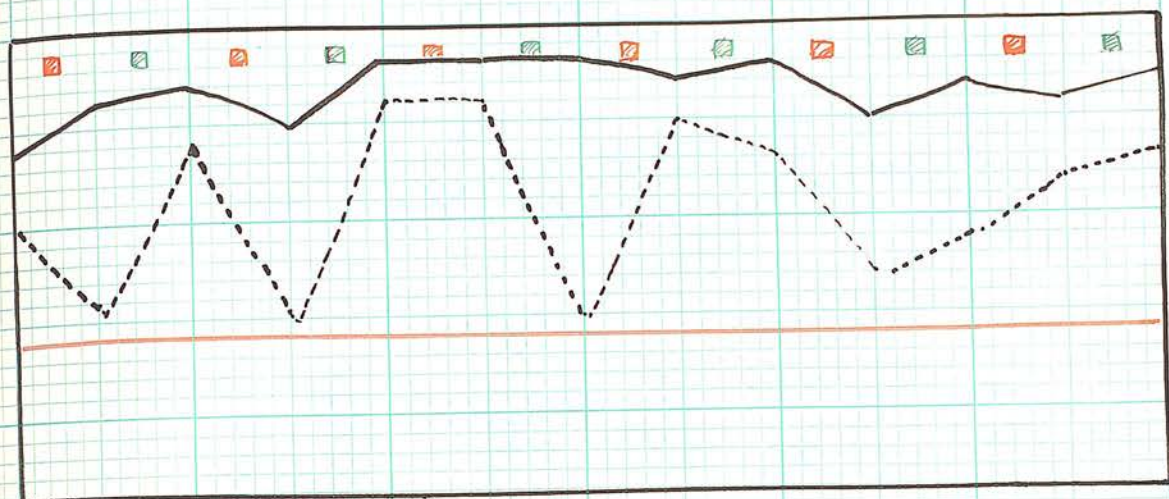
25.5.38 EJ 10 cc.

STARCH
BILE
MUCUS
BLOOD



13.6.38 (alkali) F5 wzc.

STARFISH
BILE
INJURY
BLOOD



17.6.38 FJ204.

STOMACH
BILE
MUCUS
BLOOD.

Case (3) C.

James F. (aet. 28). Miner.

Admitted: 2:6:38. Discharged: 4:7:38.

Complaint: Epigastric pain for three years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-Magsorbent.

History: During the past three years he has suffered from intermittent attacks of epigastric pain, with remissions sometimes lasting as long as six weeks. For the last three months the pain has been constant. It is burning and sharp in character and comes on about two hours after meals, and is relieved by the taking of food or alkali. It is felt in the epigastrium to the right of the midline, and sometimes in the back. There has been no vomiting. The pain does not wake him at night and is not influenced by the taking of any special types of food. Flatulence and heartburn are frequently experienced. Bowels are regular, and there has been no difference in the appearance of the motions. His appetite is very poor, and he has been losing weight.

Examination: He is a sparely built man, and lies with an anxious expression. He smokes 10-15 cigarettes per day and is a teetotaller. He is unmarried, but lives in good lodgings.

Alimentary: Teeth good: tongue dirty but moist, throat clear. Abdomen is well covered and moves well with

respiration. There is tenderness over the right rectus below the costal margin, and a slight degree of muscle rigidity is present. Splashing is not elicited. Liver and spleen not enlarged. No pain, tenderness or rigidity elsewhere.

Cardiovascular: Nil to note. BP 120/90.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "ulcer deformity of duodenal cap. No evidence of stenosis."

Urine negative for casts, albumen etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

Notes: This man is suffering from the presence of duodenal ulceration, confirmed radiologically. He improved rapidly in hospital, and tenderness and rigidity were absent after five days; no recurrence since discharge two months ago. Test meal shows a mild degree of hyperchlorhydria, and long test meal shows that there is a tendency for the acidity to rise late in the day, though this is controlled; there is further improvement two weeks later.

Conclusions: Clinically well: mild hyperchlorhydria, affected by treatment. Controlled.

Case (3) D.

James H. (aet. 22). Hotel Keeper.

Admitted: 15:7:38. Discharged: 5:8:38.

Complaint: Epigastric pain for one year.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-magsorbent.

History: During the past year he has suffered from increasing abdominal discomfort, which has settled in the epigastrium and is now a gnawing pain. He attributes this to irregular meals and excessive smoking. The pain comes on two hours after food, and is sharp in character, slightly relieved by the taking of food. Recently he vomited once or twice, and this relieved him.

Appetite is poor, and he has lost weight. He suffers a good deal from eructation and flatulence and finds that the bringing up of wind relieves the pain.

Bowels are costive, and he has lost weight recently.

Examination: He is a thin, highly strung man, who is acutely worried about his condition. No obvious osseous abnormality.

He smokes 20-30 cigarettes per day, and drinks beer or spirits as the occasion offers.

Alimentary: Top teeth false: bottom set good. Tongue dirty and moist: throat clear. Abdomen is thin, and moves freely on respiration. There is slight muscular rigidity in the upper right rectus and some tenderness on deep depression. No splashing elicited. No

enlargement of liver or spleen.

Cardiovascular: Nil to note. BP 110/86.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary. Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

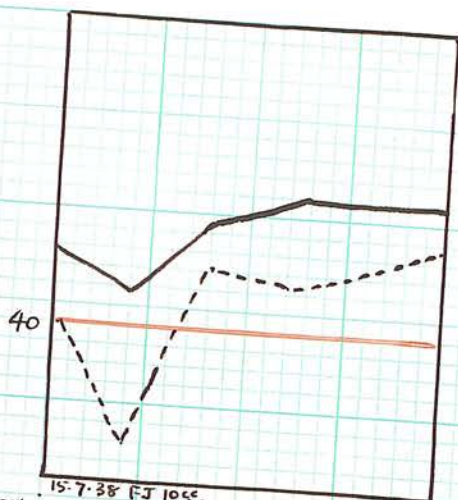
X-Ray shows scarring and deformity of duodenum: no evidence of pyloric stenosis.

Urine negative for casts, albumen etc. (done daily).

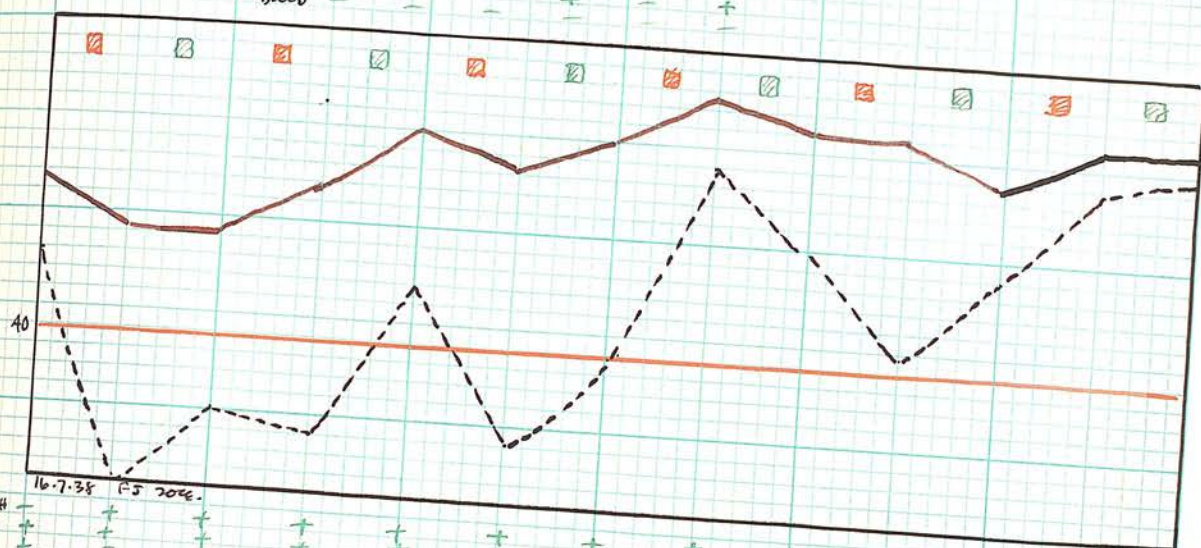
Urinary pH varied between 6.5 and 7.5.

Notes: This man is suffering from duodenal ulceration, confirmed radiologically. He did well in hospital and has had no recurrence since discharge two months ago. Test meal shows hyperchlorhydria associated with a "climbing" curve, and this hyperchlorhydria is not controlled by treatment, though there is a slight drop after the second week.

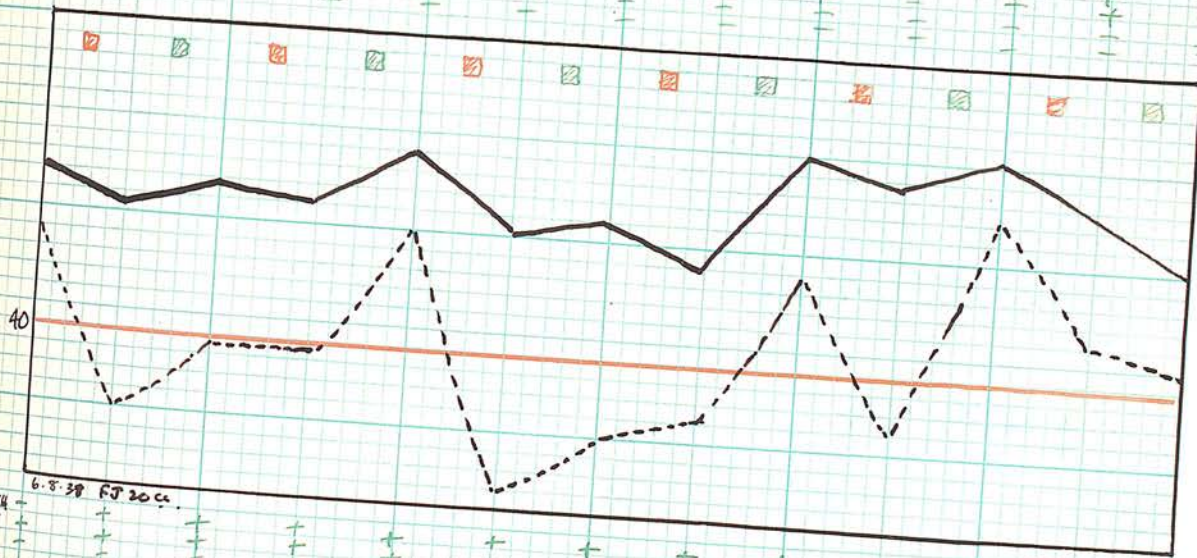
Conclusions: Clinically well: severe hyperchlorhydria, unaffected by treatment. Not controlled.



STARCH
BILE
MUCUS
BLOOD



STARCH
MUCOS
BILE
BLOOD



STARCH	-
MUCUS	+
BILE	-
BLOOD	-

Case (3) E.

John O'N. (aet. 33). Railwayman.

Admitted: 11:7:38. Discharged: 10:8:38.

Complaint: Epigastric pain for two years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-magsorbent.

History: For the past two years he has suffered from epigastric pain, which has gradually got more severe. It comes on 1½ hours after food, and is gnawing in character and sometimes felt in the back. It is not relieved by food, but is by alkali, and sometimes wakes him at night. Bowels are regular, and appetite fair. He has not lost weight. There has been no vomiting, but a good deal of heartburn and acid eructation. No flatulence.

Examination: He is a well built man, but has obviously lost some weight. No osseous abnormality.

He smokes a pipe (3 ozs. tobacco per week) and drinks beer at the week-end. He is married and has a good home.

Alimentary: Teeth false: tongue dirty: tonsils enlarged. Abdomen well covered and moves freely on respiration. There is slight tenderness on palpation in the right epigastric region and definite muscular rigidity is present. Splashing is elicited easily. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 130/96.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

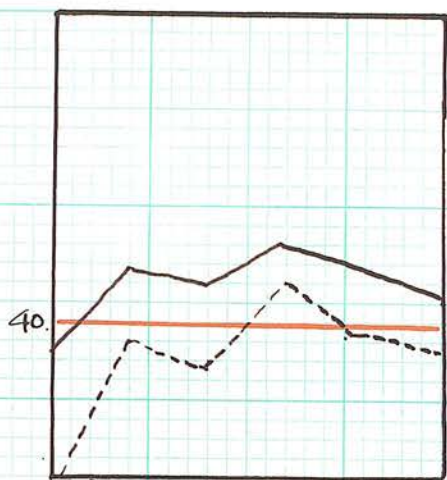
X-Ray shows "Oesophagus and stomach negative. Duodenal cap persistently deformed and niche seen - ulcer present."

Urine negative for casts, albumen etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

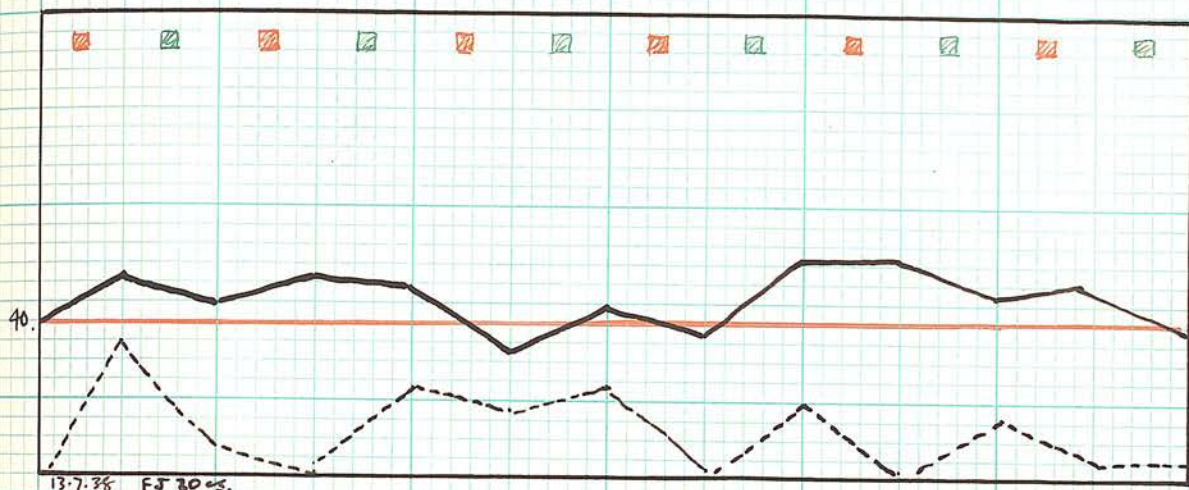
Notes: This man is suffering from the presence of a duodenal ulcer, confirmed radiologically. He improved rapidly in hospital and has had no further trouble since discharge two months ago. Test meal shows mild hyperchlorhydria, and this is markedly influenced by treatment.

Conclusions: Clinically well: mild hyperchlorhydria, responding well to treatment. Controlled.



12.7.38 FJ 30 cc.

STARCH	-	+	+	+	+	-
BILE	+	-	-	-	-	-
MUCUS	+	+	+	+	+	-
BLOOD	-	-	-	-	-	-



13.7.38 FJ 30 ч.

[illegible]

Case (3) F.

James McG. (aet. 40). Clerk.

Admitted: 20:7:38. Discharged: 16:8:38.

Complaint: Epigastric pain for six years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-magsorbent.

History: During the past six years he has suffered from epigastric pain, not definitely related to the taking of food. It has got worse and is now almost constant and is not relieved by any agents. It is felt in the midline and is "heavy and burning" and does not go through to the back. It sometimes wakes him at night. No heartburn, though flatulence occasionally troubles him. No vomiting. He has lost weight; no alteration in colour of motions, which are regular.

Examination: He does not look well, and constantly wears a frown, and speaks querulously. No osseous abnormality.

He is married and has a good home. Smokes 10-20 cigarettes daily, and is a teetotaler.

Alimentary: Teeth false, tongue dirty, throat clear. Abdomen is thin, but moves well on respiration. There is slight rigidity over the right upper rectus and tenderness on deep palpation. Splashing elicited. No enlargement of liver or spleen.

Cardiovascular: Nil to note. BP 140/106.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

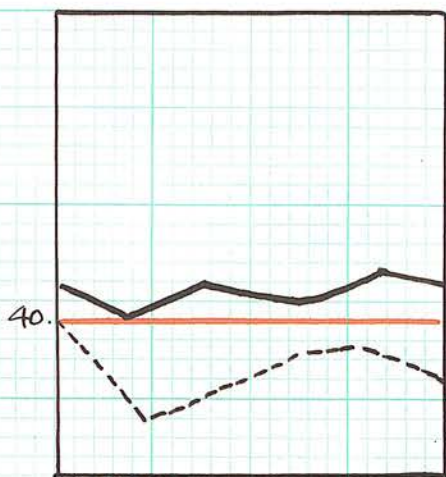
X-Ray shows "Oesphagus and stomach negative. Duodenal cap persistently deformed and difficult to fill."

Urine negative for casts. albumen etc. (done daily).

Urinary pH varies between 6.5 and 7.5.

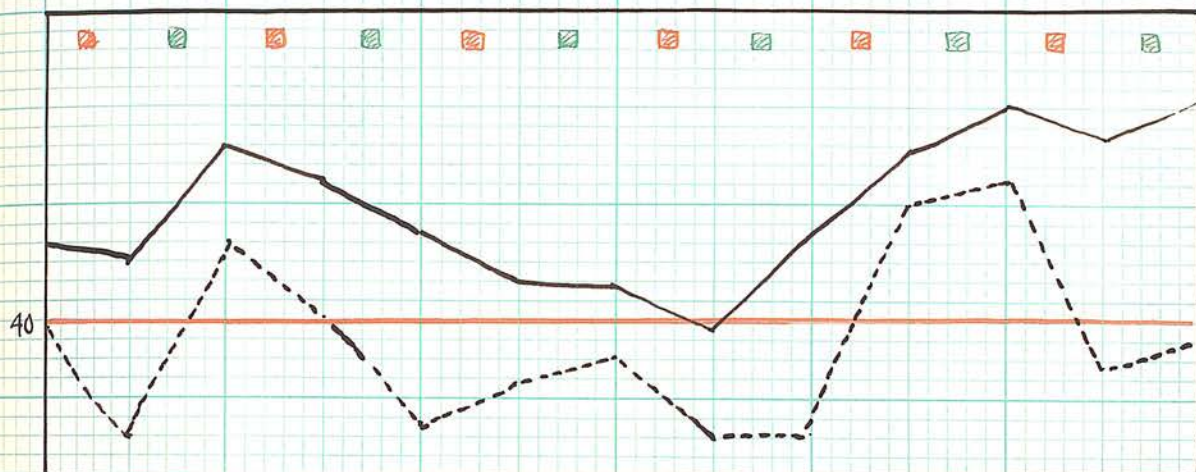
Notes: This man is suffering from duodenal ulceration, not definitely confirmed by X-Ray. He did well in hospital, and has had no further trouble since discharge two months ago. Test meal shows acidity to be within normal limits; the long meal shows a definite tendency to hyperchlorhydria which is controlled after two meals.

Conclusions: Clinically well: hyperchlorhydria responding to treatment. Controlled.

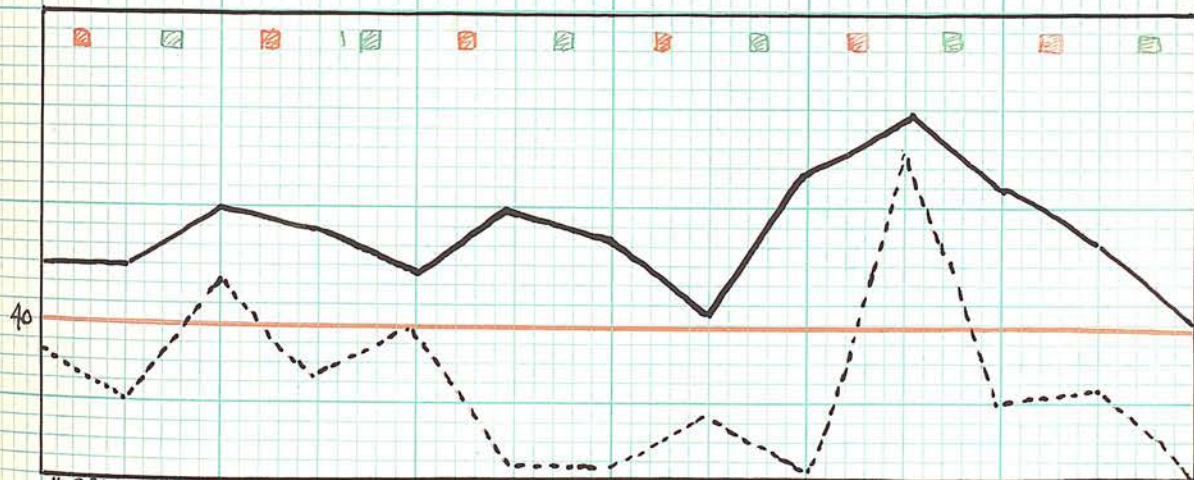


21-7-38 FJ 10cc.

	1	2	3	4	5	6
STARCH	+	+	+	+	-	-
BILE	-	-	-	-	-	-
MUCOS	+	+	-	+	-	+
BLOOD	-	-	-	-	-	-



22.7.38 FJ 40 cc.

[illegible]

14.8.38 FJ 2022

[illegible]

Case (3) G.

William S. (aet. 38). Miner.

Admitted: 12:7:38. Discharged: 14:8:38.

Complaint: Epigastric pain for three years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-Magsorbent.

History: During the past three years he has suffered from epigastric pain, sharp and burning in character and felt in the midline and occasionally the back. It comes on $1\frac{1}{2}$ - $2\frac{1}{2}$ hours after food, and is relieved by taking food or alkali, or by self-induced vomiting. Acid eructation is troublesome, but there is no flatulence. He has occasional remissions lasting from 1-7 weeks. Bowels are constipated. His appetite is poor and he has lost some weight. The pain never wakes him at night.

Examination: He is a thin, poorly nourished man, and looks worried and anxious. No osseous deformity.

He is married, and has a poor home. He smokes a pipe (2-3 ozs. tobacco per week) and drinks beer or spirits.

Alimentary: Teeth very bad: tongue dirty: throat clear. Abdomen is thin, but moves well on respiration. There is slight tenderness in the right epigastric region, associated with some degree of muscle rigidity. Splashing is elicited easily. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 138/110.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Stomach and oesophagus normal. Scarring of duodenal cap with slight degree of gastric retention."

Urine negative for casts, albumen etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

Notes: This man is suffering from duodenal ulceration, confirmed radiologically. There was a rapid improvement in hospital, and no recurrence of symptoms since discharge two months ago. Test meal shows hyperchlorhydria: this is markedly modified by treatment, but shows a tendency to recur again later.

Conclusions: Clinically well: hyperchlorhydria modified by treatment. Controlled.

Case (3) H.

James McK. (aet. 32). Lorry Driver.

Admitted: 16:6:38. Discharged: 11:7:38.

Complaint: Epigastric pain for seven years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-Magsorbent.

History: For the past seven years he has suffered from epigastric pain, with occasional remissions lasting from 1-3 months. The pain is gnawing in character, comes on about 2 hours after food, and is relieved by the taking of food or alkali. The pain has recently become more persistent and severe, and he has had attacks of vomiting in the last three months. No flatulence or acid eructation. The bowels are constipated and he has lost weight.

Examination: He is a tall, powerful man, but thin for his build, and hollow-cheeked. No osseous abnormality.

He smokes a pipe (3-4 ozs. tobacco per week) and drinks one pint of beer per day. He is married and has a good home.

Alimentary: Teeth false: tongue dirty: throat clear.

Abdomen is thin and moves freely on respiration.

There is some tenderness in the upper right quadrant of the abdomen, but no muscular rigidity. Splashing is not elicited. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 128/88.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

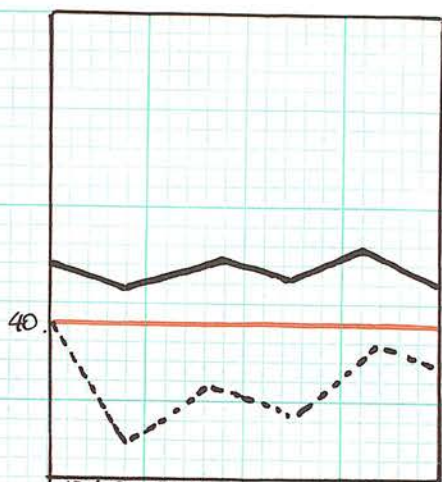
X-Ray shows "Stomach and oesophagus negative. Duodenum shows presence of ulcer crater without organic obstruction."

Urine negative for casts, albumen etc. (done daily).

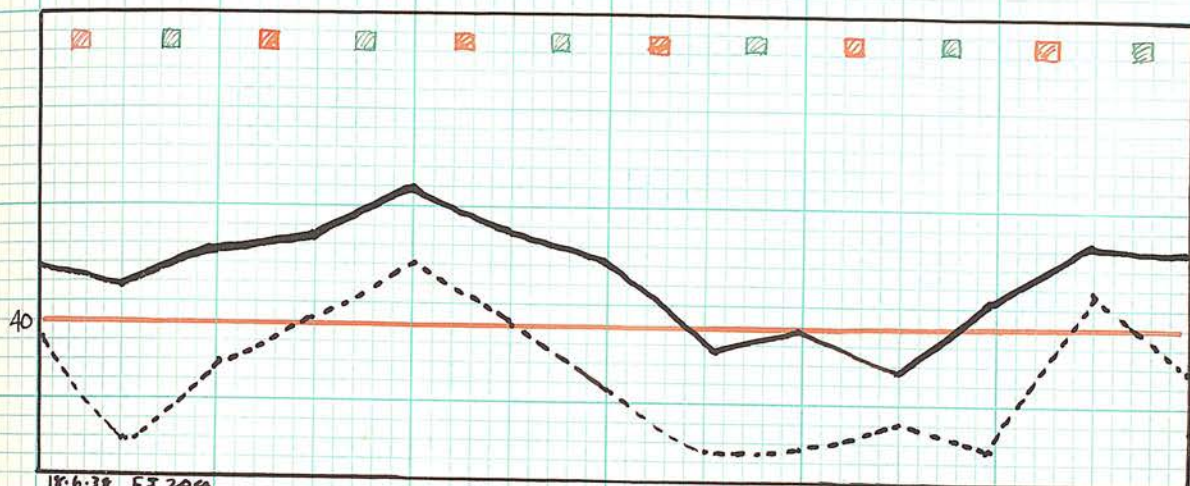
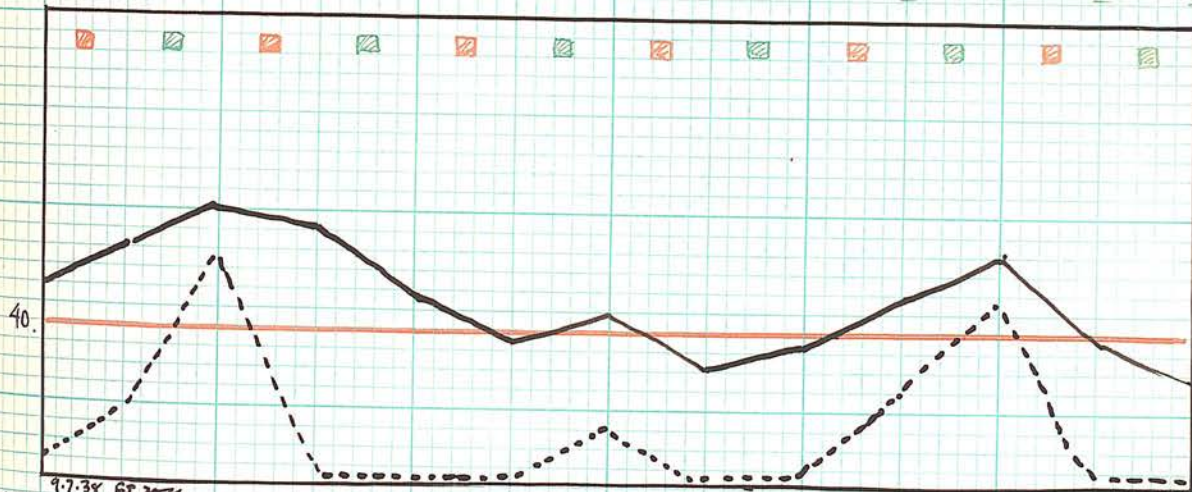
Urinary pH varied between 6.5 and 7.5.

Notes: This man is suffering from the presence of a duodenal ulcer, confirmed radiologically. He did well in hospital and has had no further trouble since discharge, two months ago. Test meal shows acidity to be within normal limits, but long test meal shows a tendency for acidity to rise, and the acidity is much more modified after two weeks.

Conclusions: Clinically well; mild hyperchlorhydria modified by treatment, and further modified after two meals. Controlled.



	17.0.58	15 SEP.				
STARCH	-	+	+	+	+	-
BILE	-	-	-	-	-	-
MUCUS	+	+	-	+	-	+
BLOOD	-	-	-	+	+	-

[illegible][illegible]

Case (3) I.

Richard L. (aet. 22). Shop Assistant.

Admitted: 4:7:38. Discharged: 6:8:38.

Complaint: Epigastric pain for one year.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-Magsorbent.

History: For the past year he has been troubled by a feeling of epigastric discomfort after meals, which has lately become actually painful. It comes on about two hours after meals and lasts until food is taken. It sometimes wakes him at night. He has a fairly good appetite, and has had no heartburn, and no vomiting. He has lost a little weight. Bowels tend to be constipated, and there has been no change in the colour of the motions. The pain is sharp in character, but is never felt in the back.

Examination: He is a sparely built man, rather nervous, and does not seem ill. No osseous abnormality.

He smokes about 10 cigarettes per day, and is a teetotaler. He is unmarried.

Alimentary: Tongue dirty: teeth carious: throat clear. Abdomen is well covered and moves freely on respiration. There is tenderness on palpation in the epigastrium just to the right of the midline, and a degree of muscle rigidity is associated with this. Splashing is easily elicited. Liver and spleen are not enlarged.

Cardiovascular: Nil to note.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

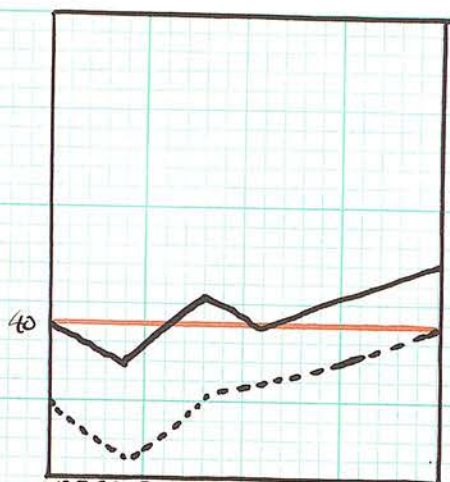
X-Ray shows "Scarring and deformity of duodenum consistent with presence of ulcer."

Urine negative for casts, albumen etc. (done daily).

Urinary pH varied between 6.5 and 7.5.

Notes: This man is suffering from duodenal ulceration, confirmed radiologically. There was rapid improvement in hospital, and no further trouble since discharge two months ago. Test meal shows mild hyperchlorhydria, and this is a satisfactory maintenance of a low acidity, with only one major rise per day during treatment.

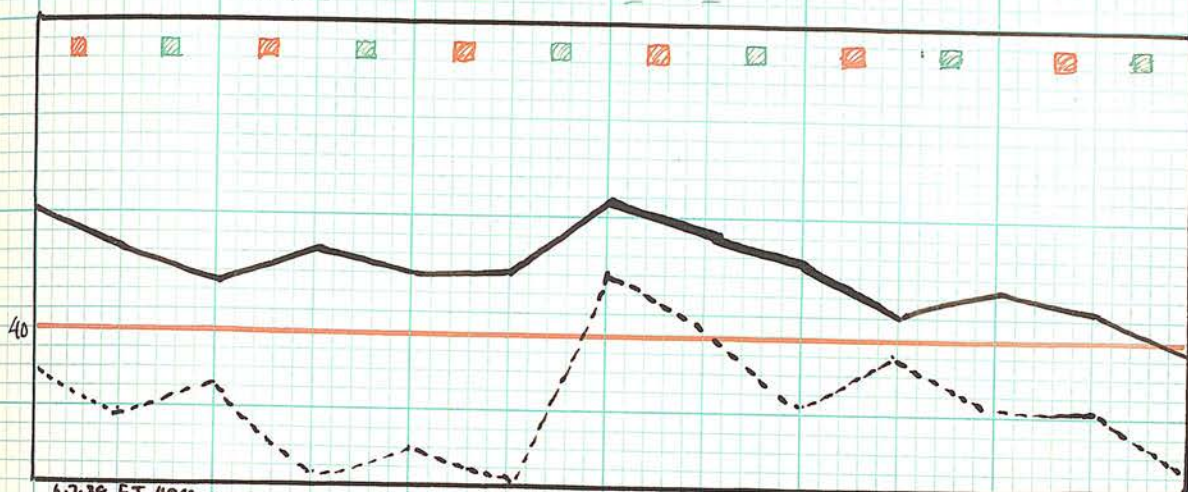
Conclusions: Clinically well: mild hyperchlorhydria, responding to treatment. Controlled.



5.7.38 FJ 204.

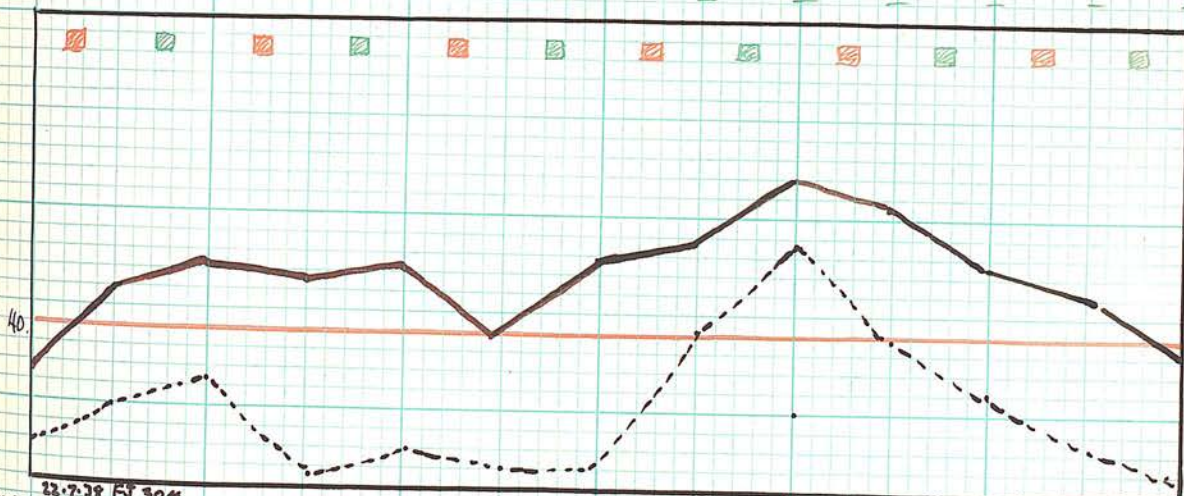
STARCH
BILE
MUCUS
BLOOD

STARCH	+	+	+	+	-	-
BILE	+	-	-	+	-	-
MUCOS	+	+	-	+	+	+
BLOOD	-	-	-	-	-	-



6.7.38 FJ 404.

STARCH -
BILE -
MUCUS +
BLOOD -

[illegible]

22.7.78 FJ 304.

STACH
OLE
RUCUS
BLOND.

[illegible]

Case (3) J.

Elizabeth F. (28). Housemaid.

Admitted: 11:7:38. Discharged: 14:8:38.

Complaint: Epigastric pain for four years.

Diagnosis: Duodenal ulcer.

Treatment: Sippy-Magsorbent.

History: For the past four years she has suffered from abdominal pain, with occasional remissions lasting from 1-3 months. The pain is dragging in character, and comes on $1\frac{1}{2}$ -2 hours after food and is relieved by the taking of food or alkali. It is sometimes felt in the back. She has lost weight. Her appetite is poor and the bowels are costive. The pain occasionally wakes her at night. There has been no vomiting, but she has been troubled by heartburn and flatulence. Menstrual periods are regular, but scantier than previously.

Examination: She is a highly strung girl, anxious looking and rather thin. Hair is sparse and dry. No osseous deformity.

She is a teetotaller, and smokes five to seven cigarettes per day.

Alimentary: Teeth carious in top set, good lower set. Tongue dirty, throat clear. Abdomen thin but well formed and moves freely on respiration. There is slight tenderness in the midline in the epigastrium and some rigidity of both upper recti. Splashing is not elicited. Liver and spleen not enlarged.

Cardiovascular: Nil to note. BP 110/74.

Respiratory: Nil to note.

Nervous: Nil to note.

Genitourinary: Nil to note.

Wassermann reaction negative.

Benzidine reaction of stools negative (done every second day).

X-Ray shows "Oesophagus and stomach normal. Duodenal cap persistently deformed and a niche seen - duodenal ulcer."

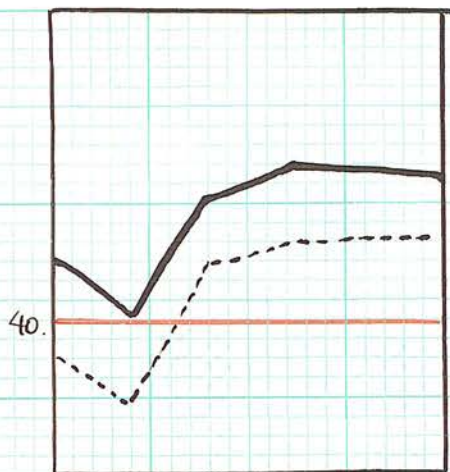
Urine negative for casts, albumen etc. (except during menstruation).

Urinary pH varied between 6.5 and 7.5.

Notes: This woman is suffering from the presence of duodenal ulceration, confirmed by X-Rays.

Improvement was rapid in hospital and no further trouble has been experienced since discharge two months ago. Test meal shows severe hyperchlorhydria which is not much affected at first by treatment: after two weeks, however, it is satisfactorily controlled.

Conclusions: Clinically well: severe hyperchlorhydria markedly modified by treatment. Controlled.



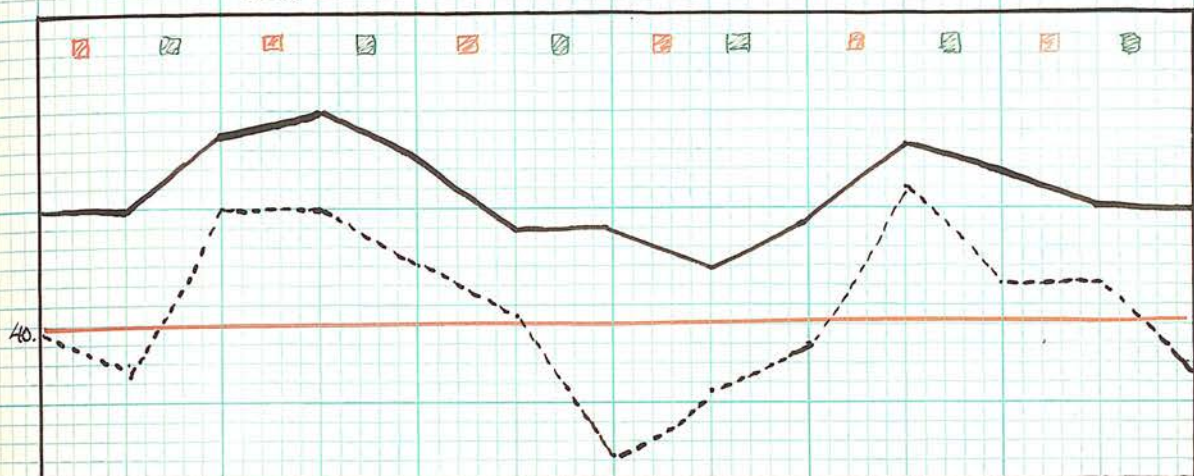
12-7-28 F J 40 cc.

STARCH

BILE

ηθικός

BLOTT



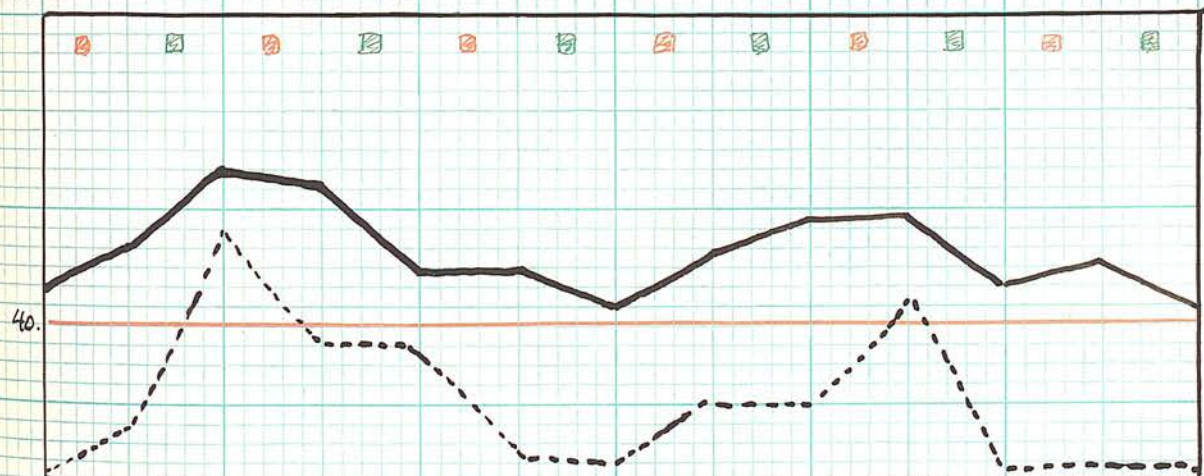
13.7.39 FJ 304.

STARCH

FILE

fOCUS

BLOOD.



29.7.34 FJ Soc.

STADT

BILE

NUCUS
K. 100

2. 2. 2.

Case (4) A.

Mrs. Mary C. (aet. 42). Housewife.

Admitted: 12:4:38. Discharged: 17:5:38.

Complaint: Epigastric pain for eight years.

Diagnosis: Pyloric stenosis secondary to duodenal ulcer.

Treatment: Gastro-enterostomy.

History: She has had "Stomach trouble" for eight years, which has not responded to medical treatment. The epigastric pain comes on about one hour after food, is sharp in character and not relieved by food or alkali. It has been persistent for the last six months. There has been daily vomiting for the last three months. She has lost a lot of weight.

Examination: She is thin, spare woman, obviously much emaciated.

On palpation of the abdomen, there is marked tenderness in the midline in the epigastrium, and some muscle boarding. Splashing is easily elicited.

Examination of other systems is negative.

X-Ray reveals the presence of a severely scarred and deformed duodenal cap, and a definite hold-up of gastric contents.

Wassermann reaction negative.

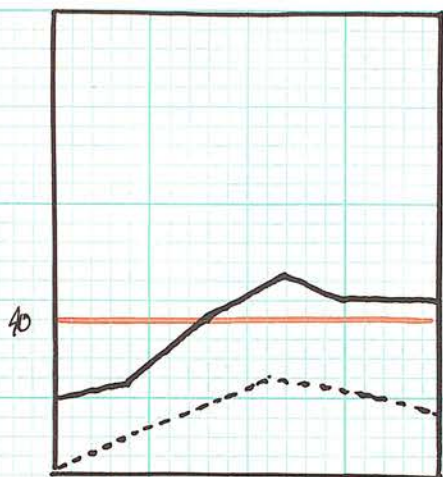
Benzidine reaction of stools negative.

At operation (22:4:38) the scarring of the pylorus and pyloric stenosis was demonstrated. A gastro-

enterostomy was performed, and a small fibrotic appendix removed.

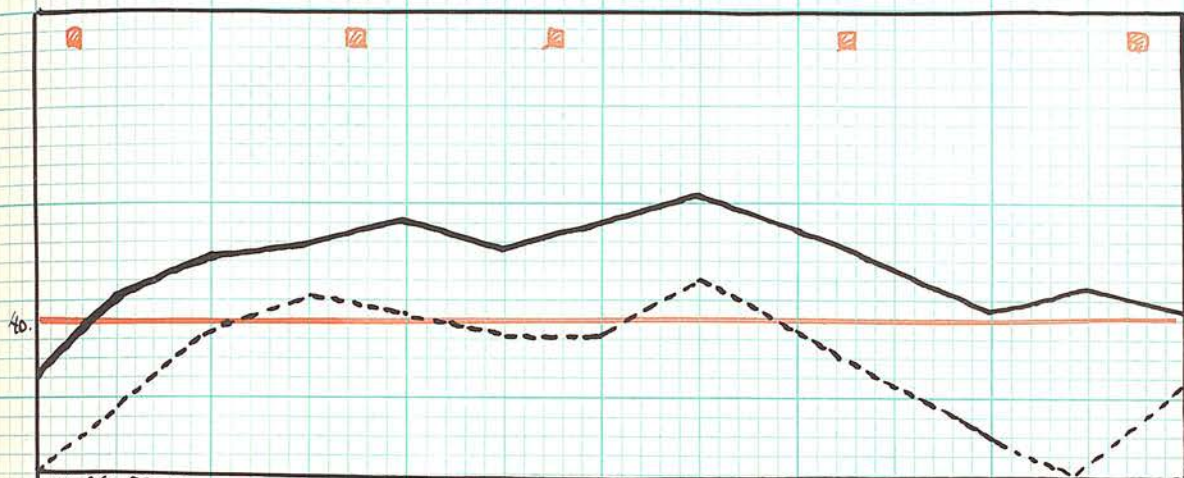
Notes: This woman demonstrated that pyloric stenosis, clinically obvious, can be present without marked hyperchlorhydria. Following operation, the acidity did not drop at once, as might be expected, but took some months to adjust itself to a lower level.

Mailer (personal communication) considers that not uncommonly the acidity may rise later to its previous level.

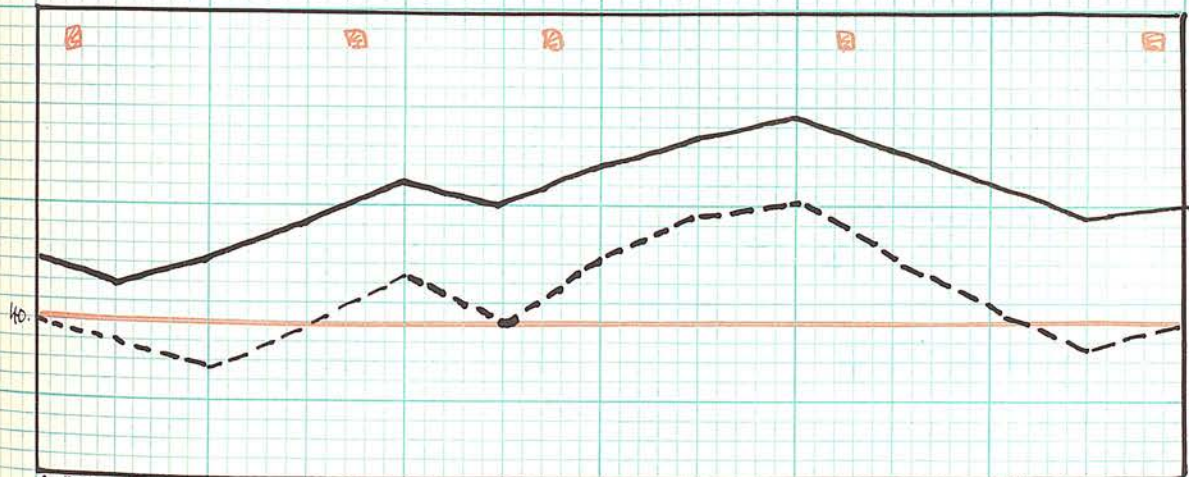


13.4.38 73 бусе.

STAR RCH	+	+	+	+	+
BILE	-	-	-	-	-
MUCOS	-	+	+	+	+
BLOOD	-	-	-	-	-

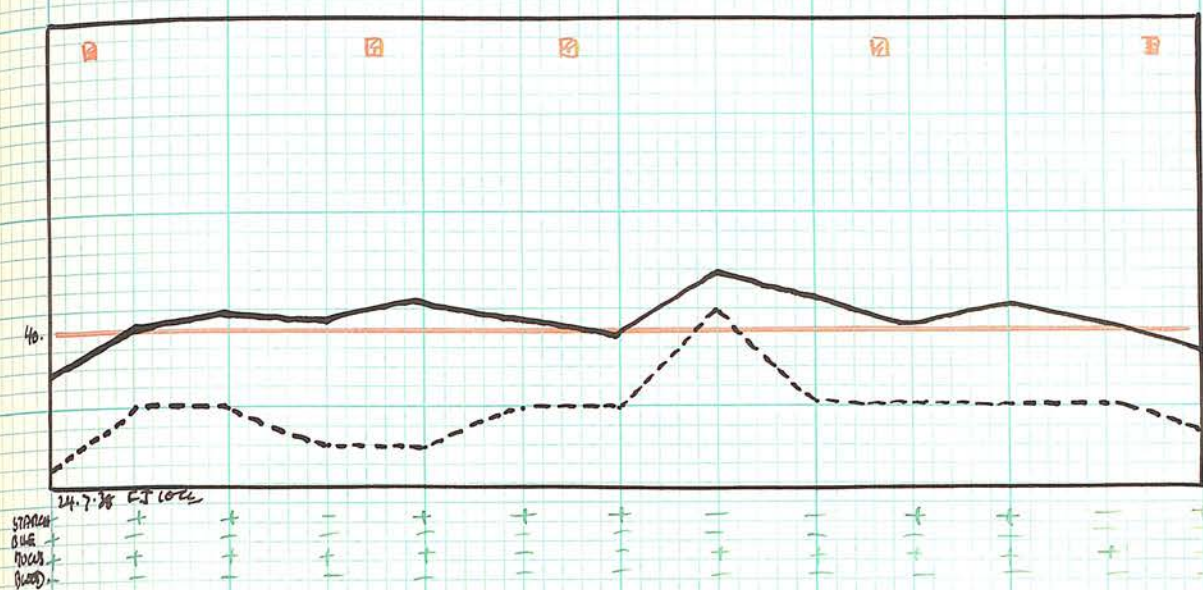


18.4.38 7560cc.

[illegible]

20.5.36 FJ MAR.

	10.5.36	27. Mar.										
Streck:	+	+	+	—	+	+	+	+	—	+	—	+
Öffn.	+	+	—	—	+	+	+	+	—	+	—	+
Neuig.	+	+	—	—	+	+	+	+	—	+	—	+
Reind.	+	+	—	—	+	+	+	+	—	+	—	+



Case (4) B.

Duncan L. (aet. 55). Labourer.

Admitted: 1:4:38. Discharged: 20:5:38.

Complaint: Epigastric pain for four years.

Diagnosis: Duodenal ulcer with pyloric stenosis.

Treatment: Gastro-enterostomy.

History: Each spring for the last four years he has suffered from attacks of epigastric pain, accompanied by flatulence, nausea and vomiting. It comes on about two hours after meals and is not relieved by taking food or alkali. The last attack has lasted three months and is getting gradually worse as the pain is now almost constant and vomiting more frequent and copious.

Examination: He is a thin, rather wasted man.

On palpation of the abdomen, there is tenderness and muscle rigidity in the right epigastric region, and splashing is easily elicited. Examination of other systems reveals no abnormality. X-Ray shows "Stomach and oesophagus normal. Duodenal cap persistently deformed, and two niches seen."

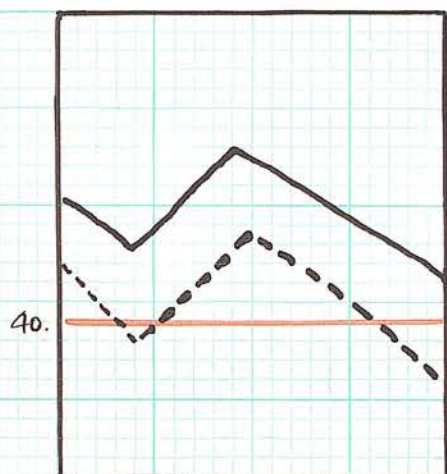
Wassermann reaction negative.

Benzidine reaction of stools negative.

Operation (15:4:38) revealed the presence of a large active duodenal ulcer and some degree of pyloric stenosis. A gastro-enterostomy was performed,

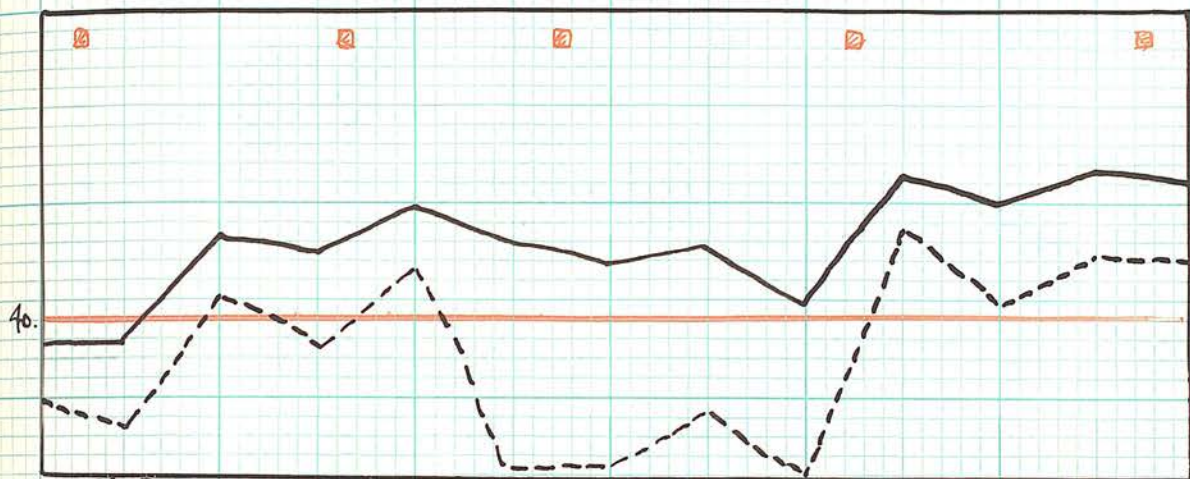
and an atrophic appendix removed at the same time.

Notes: This case again shows that the types of graphs seen in cases of ulcer and stenosis are markedly modified following operation. The clinical findings were verified at operation.

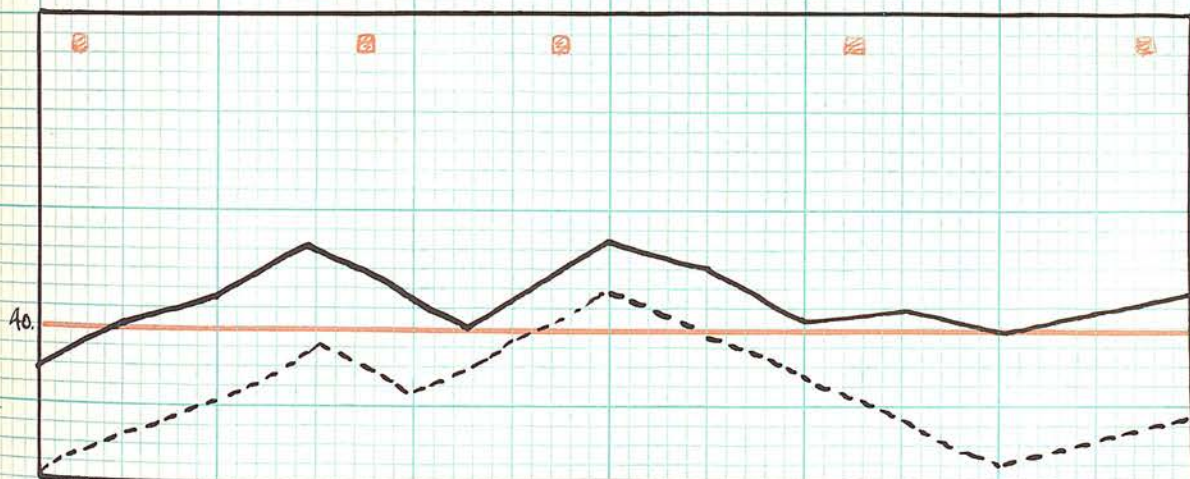


4.4.38 FJ 40cc.

STARCH	+	+	+	+	+
BILE	-	-	-	-	-
MUCOS	+	+	+	+	+
BLOOD	-	-	-	-	-



12.4.38 FJ60Cc

[illegible]

16.5.38 RJ10CC

[illegible]

Case (4) C.

Andrew D. (aet. 26). Miner.

Admitted: 12:4:38. Discharged: 10:5:38.

Complaint: Epigastric pain for two years.

Diagnosis: Duodenal ulcer.

Treatment: Gastro-enterostomy.

History: For the past two years he has been troubled by epigastric pain, coming on 1½-2 hours after food, very sharp in character and not relieved by taking food or alkali or by vomiting. It has got gradually worse and recently has been almost constantly present. He has lost a good deal of weight.

Examination: He is a spare individual of more than average intelligence and obviously worried about his condition.

On palpation of the abdomen, there is no tenderness, but there is rigidity of the upper right rectus. Splashing is not elicited. Examination of other systems is negative.

X-Ray shows "Stomach and oesophagus normal.

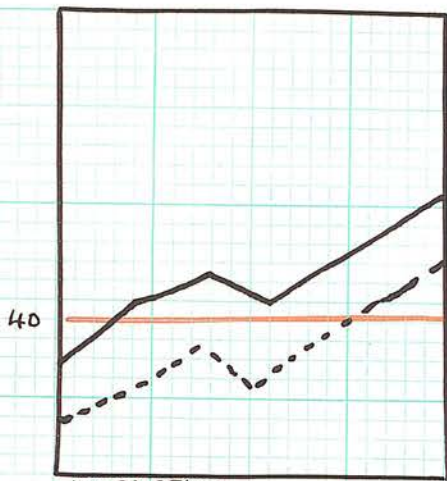
There is an ulcer crater on the first part of the duodenum, and some degree of duodenal ileus."

Wassermann reaction negative.

Operation (19:4:38) revealed the presence of an active ulcer on the first part of the duodenum, posteriously attached to the pancreas, and

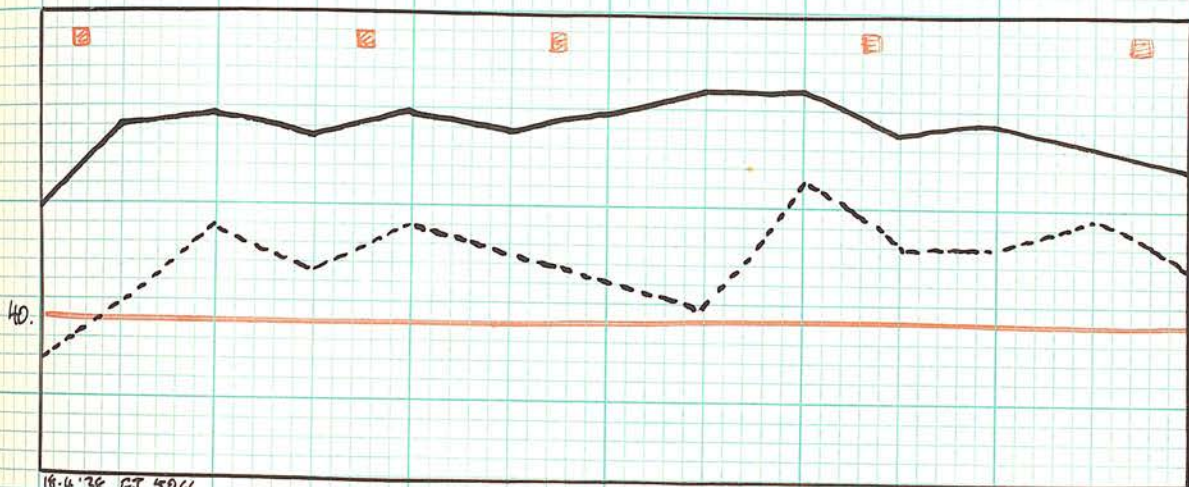
scarring and early stenosis. A gastro-enterostomy and appendicectomy was performed.

Notes: In this case, the findings predicted on clinical and biochemical grounds were verified at operation.



14.4.38 FJ60cc.

STARCH	+	+	+	+	+
BILE	-	-	-	-	-
MUCUS	-	+	-	+	+
BLOOD	-	-	-	-	-



18-4-38 FT 50CC

[illegible]

Case (4) D.

James D. (aet. 32). Miner.

Admitted: 2:12:37. Discharged: 20:2:38.

Complaint: Epigastric pain for eleven years.

Diagnosis: Chronic appendicitis: pylorospasm.

Treatment: Appendicectomy.

History: For eleven years he has been troubled by intermittent attacks of epigastric pain, coming on when he was hungry, and relieved by the taking of food or alkali. He has also suffered from flatulence, acid eructations, and nausea. There has been no vomiting or loss of weight. Bowels are constipated.

Examination: He is a well built, strong man, with neurotic tendencies.

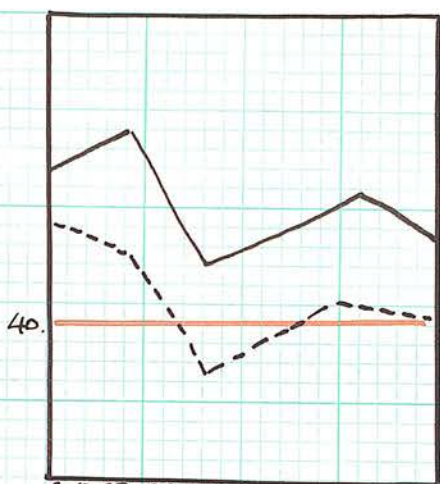
Palpation of the abdomen reveal slight tenderness but no rigidity in the epigastrium, but definite tenderness and muscle boarding over McBurney's point. Other systems negative.

Wassermann reaction negative.

Benzidine reaction of stools negative.

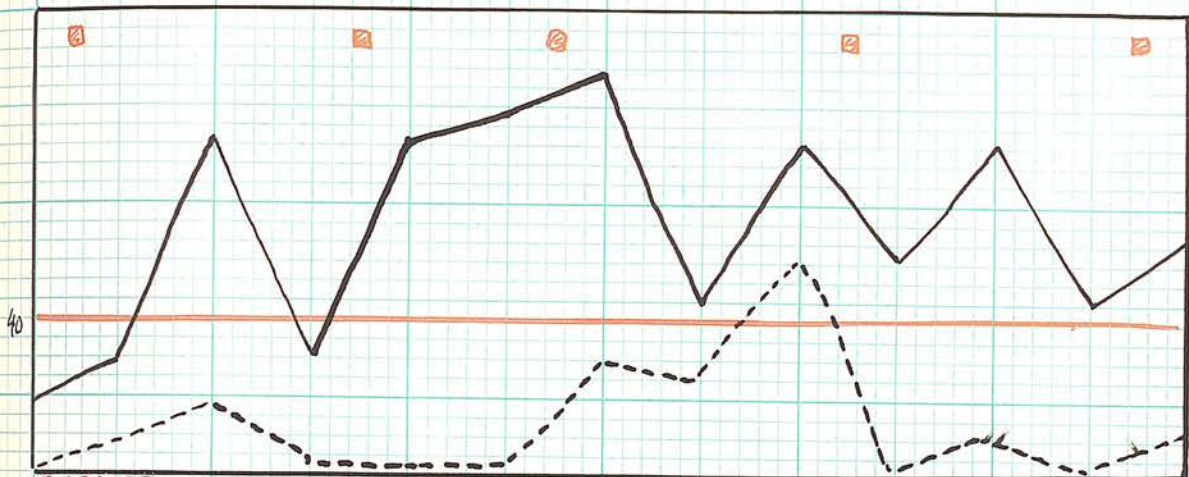
Operation (1:2:38) revealed the presence of a chronically inflamed appendix. There was no gastric or duodenal lesion, but some degree of pylorospasm was detected. Appendicectomy was performed.

Notes: This case showed the presence of hyperchlorhydria associated with an alimentary non-gastric lesion, namely appendicitis, and the hyperchlorhydria settled down after appendicectomy.

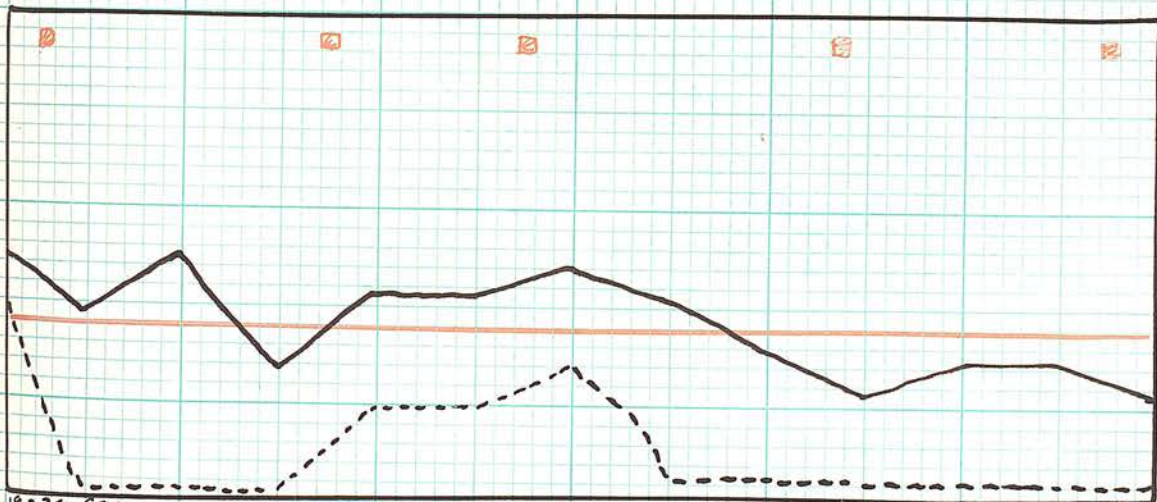


3.12.37. FJ 20cc

	5.12.57.	13.12.57.	14.12.57.	15.12.57.	16.12.57.	17.12.57.
STARCH	—	+	+	+	+	+
BILE	—	—	—	—	—	—
NUCUS	+	+	+	+	+	+
BLOOD	—	—	—	—	—	—



12.1.38 FJ 40cc

[illegible]

18.2.38 FS 1022

[illegible]

Case (4) E.

William B. (aet. 32). Labourer.

Admitted: 12:4:38. Discharged: 14:5:38.

Complaint: Epigastric pain for one year.

Diagnosis: Chronic appendicitis with pylorospasm.

Treatment: Appendicectomy.

History: For about a year he has been troubled by pain in the epigastrium, sometimes shifting to the right iliac fossa; it usually comes on about two hours after food, and is relieved by self-induced vomiting. "It is not terribly sore, but very uncomfortable." Bowels are constipated. No loss of weight.

Examination: He is a well-built man, and not unduly affected by the disorder.

Palpation of the abdomen reveals no tenderness or rigidity in the epigastrium region, but definite tenderness and a slight degree of rigidity over McBurney's point. Examination of other systems is negative.

X-Ray shows "Stomach and oesophagus normal.

Duodenal cap fills well and empties normally.

No deformity or scarring.

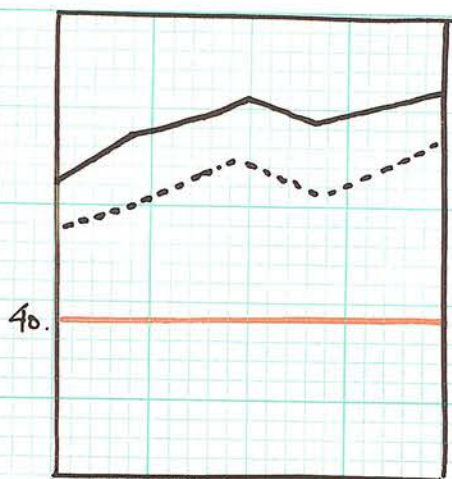
Wassermann reaction negative.

Benzidine reaction of stools negative.

Operation (20:4:38) revealed the presence of a chronically inflamed appendix, bound down by

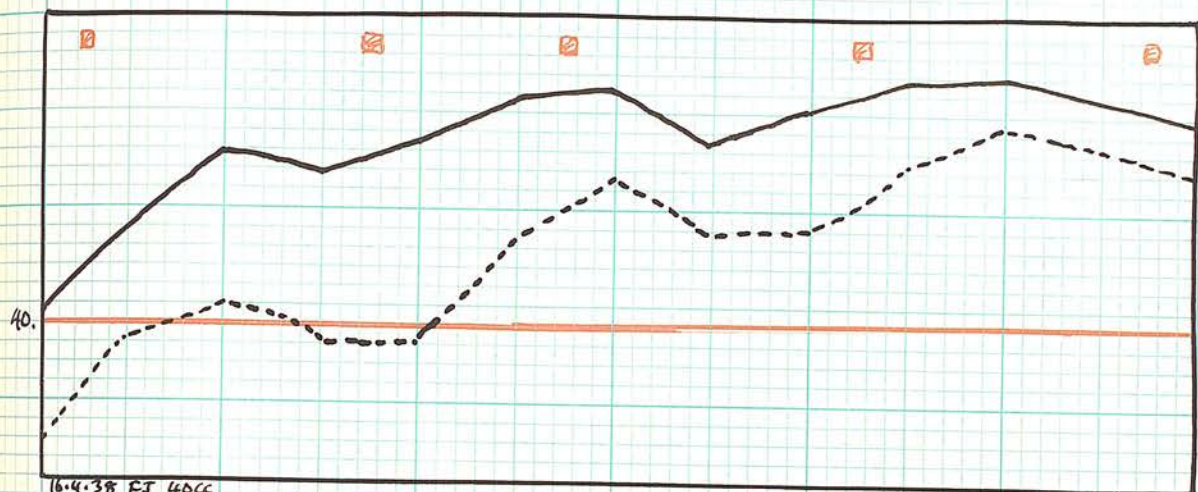
adhesions. There was no evidence of gastric or duodenal lesion. The appendix was removed.

Notes: This case also demonstrates the presence of hyperchlorhydria associated with appendicitis, but the acidity has not diminished following operation, and clinically the possibility of nervous stimulation is prominent.

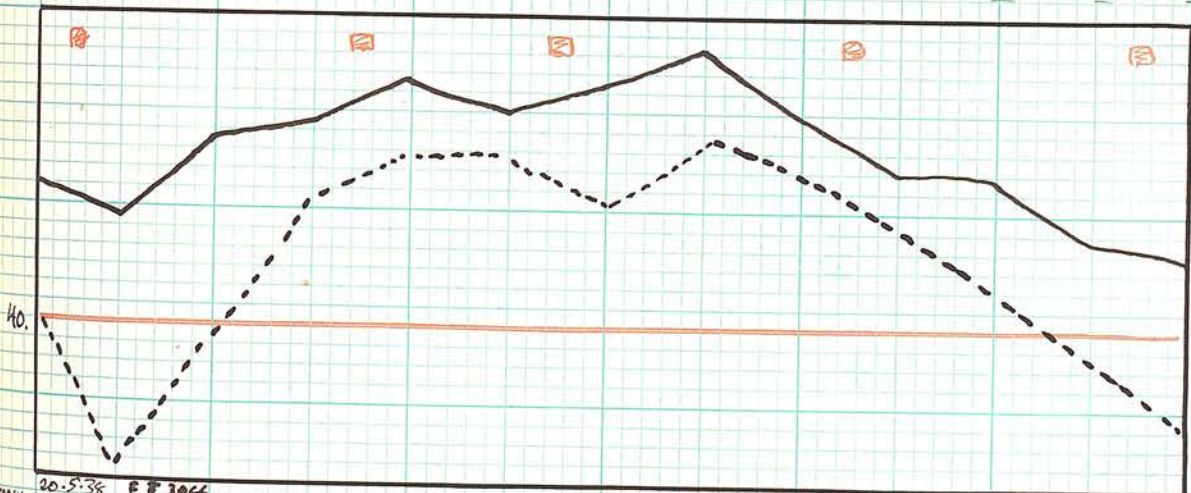


12.4.38 FJ 40cc.

STARON	+	+	+	+	+	+
BILE	-	-	-	-	-	-
MUCOS	+	+	+	+	-	+
BLOOD	-	-	-	-	-	-



16.4.38 EJ 40CC

[illegible]

20.5.38 FF 30cc

[illegible]

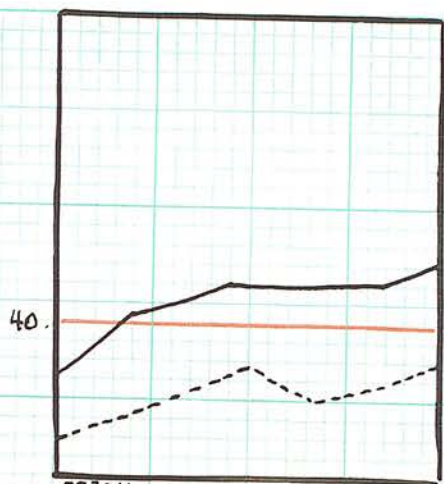
Control X. 1.

Ina McL. (aet. 32). Housewife.

This woman is well now, having convalesced after an attack of acute bronchitis, and her gastric function is taken as being normal.

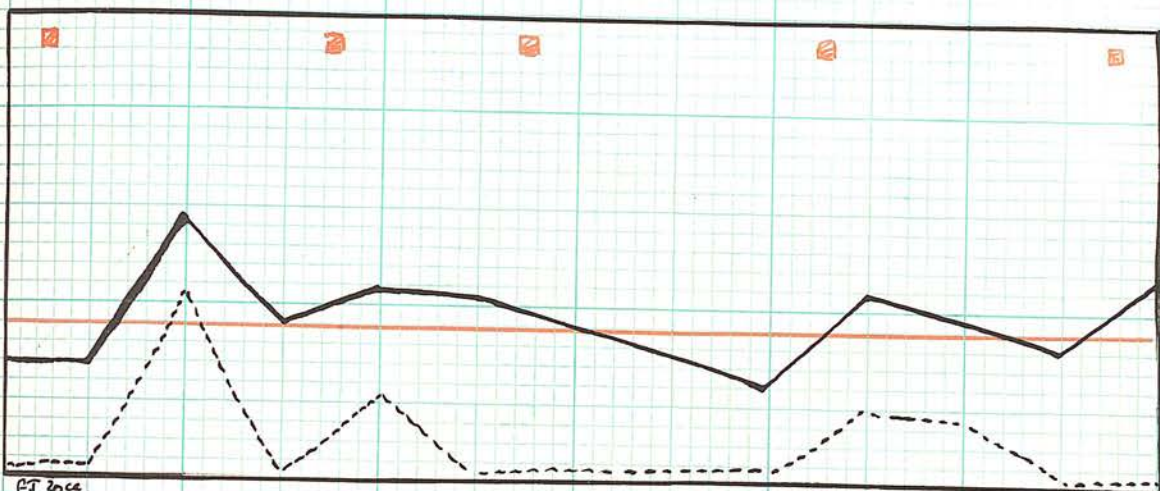
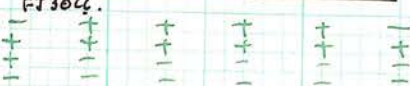
She is on ordinary diet, and has had no illnesses or gastric complaints.

Notes: It can be seen from the long test meal that in the normal person the acidity may exceed 40, but this only happens here at one time during the day: otherwise the acidity is very low, and it is possible that the one may rise may have been due to some external stimulus acting. (e.g. nausea).



FT 3044.

STARCH
MUCUS
BILE
BLOOD.



FT 3044.

STARCH
BILE
MUCUS
BLOOD.

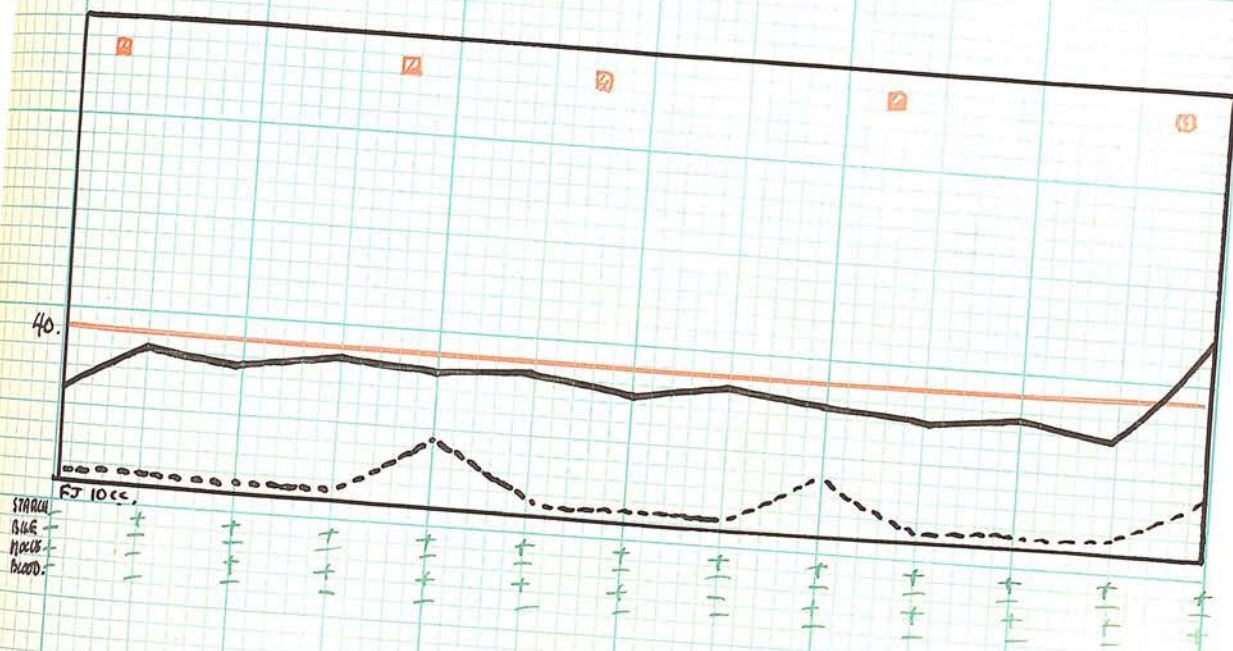
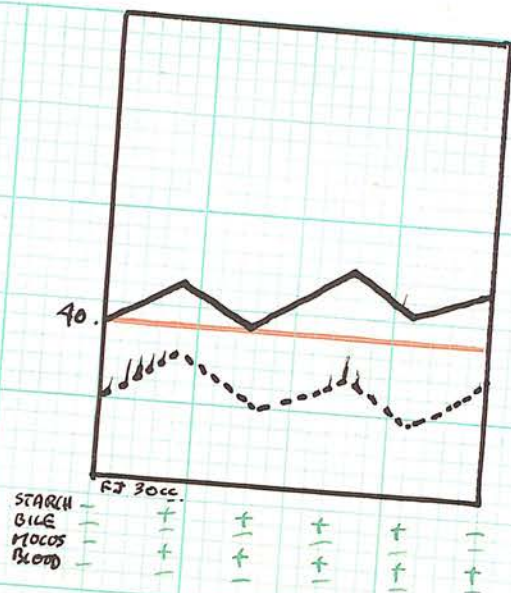


Control X. 2.

Jean McK. (aet. 38). Post Office Assistant.

This woman is well after convalescing from acute lobar pneumonia, and her gastric function is taken to be normal. She has had no illnesses and no gastric complaints, and is on ordinary diet.

Notes: This is the type of long test meal that one would suspect probably obtains in most normal people.



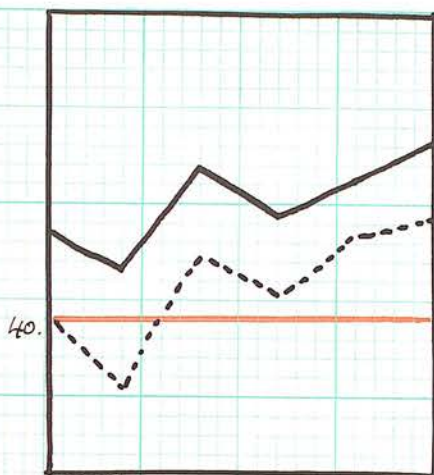
Control X. 3.

Mary McB. (aet. 18). Unemployed.

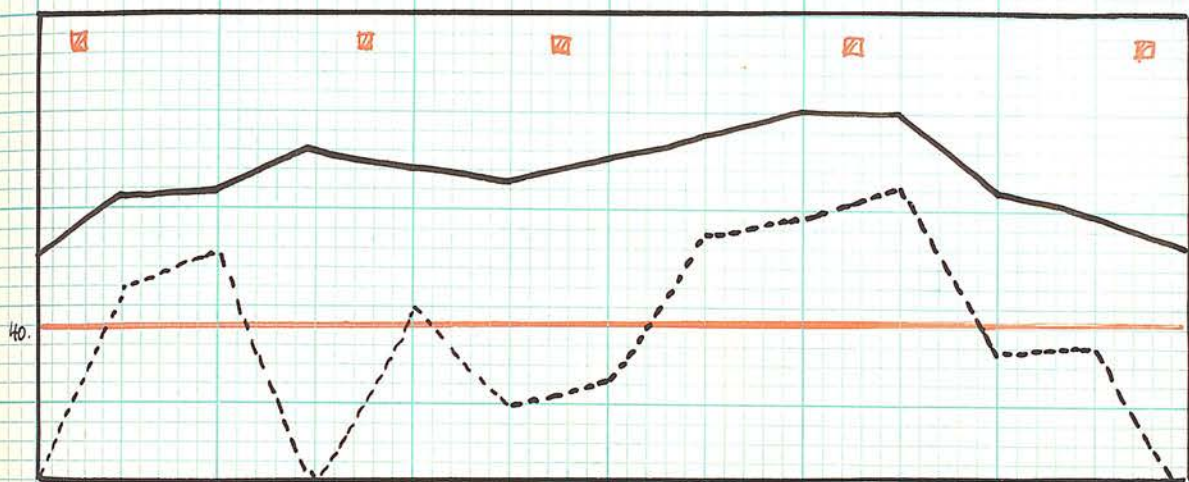
This girl is well after an attack of acute rheumatism, and her gastric function is taken as normal. She has had no previous illnesses and no gastric complaints, and is on ordinary diet.

Notes: This case is included to demonstrate the amazing variation in acidity that may occur from hour to hour in the (presumably) normal person. The severe hyperchlorhydria present in this case was not associated with any symptoms.

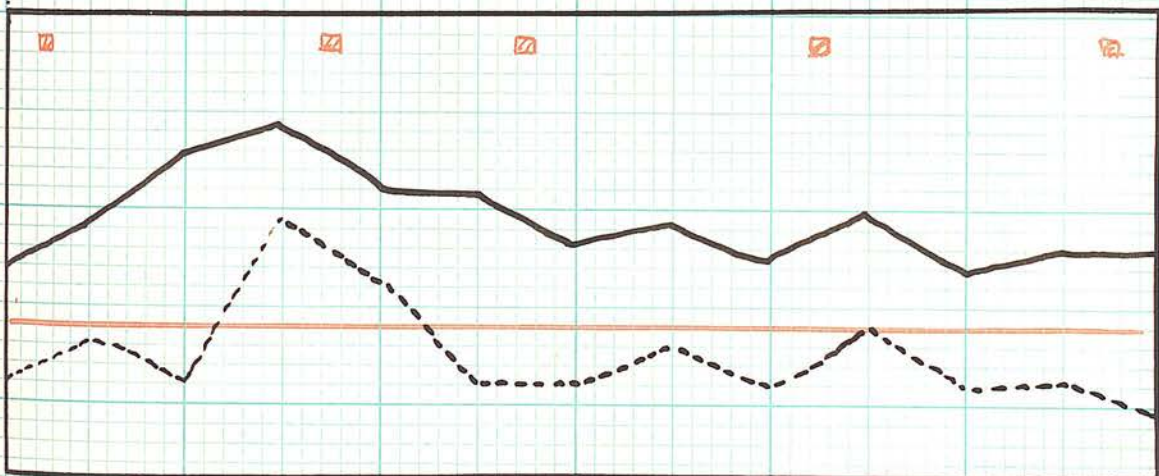
This should be compared with cases X1 and X2.



STARCH +
BILE -
MUCUS +
BLOOD -



STARCH +
BILE -
MUCUS +
BLOOD -



STARCH +
BILE -
MUCUS +
BLOOD -

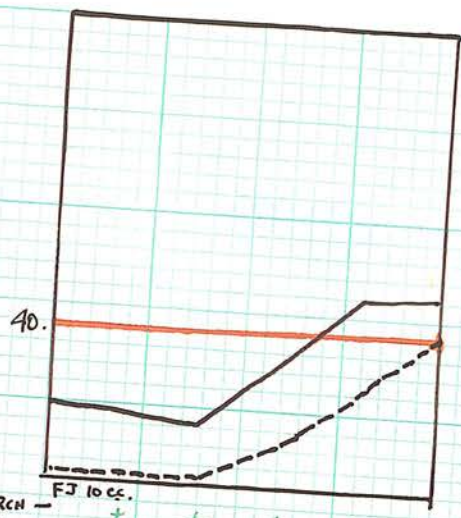
Control Y. 1.

Mrs. McN. (aet. 29). Factory Worker.

This woman is well after an attack of acute rheumatism. Her gastric function is presumed normal and was investigated by (1) an ordinary test meal and (2) gastric analysis after being started on a Sippy-alkali regime.

There is no history of any gastric trouble.

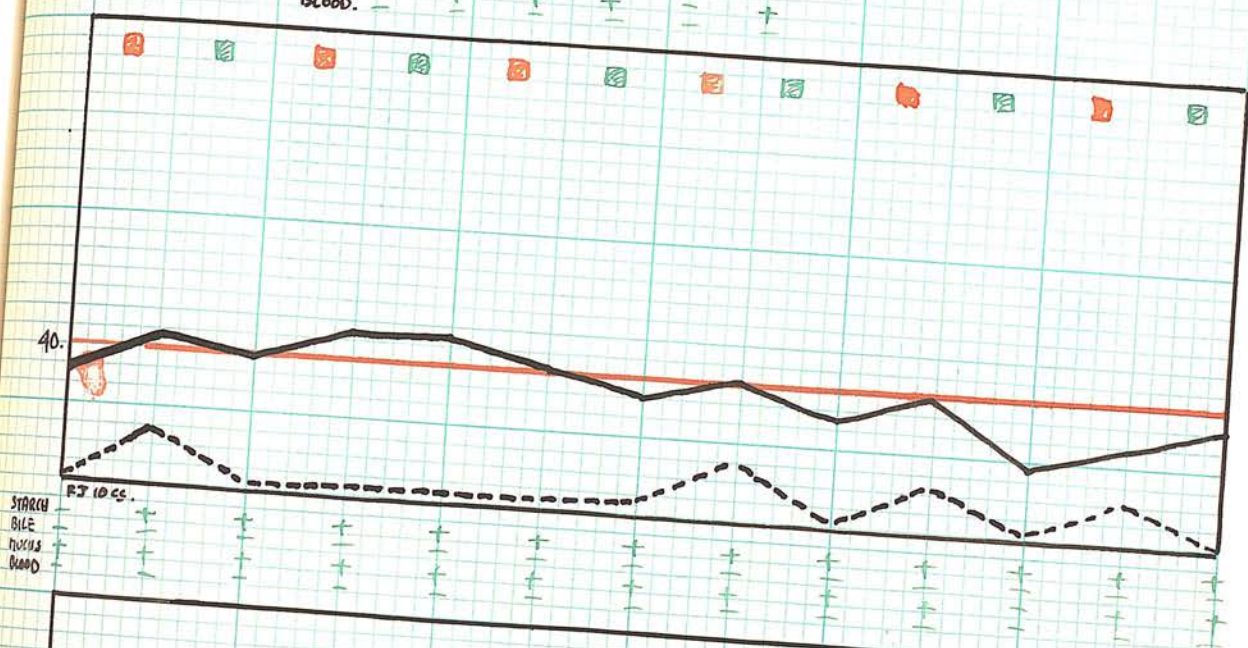
Notes: It will be seen from this case that total achlorhydria is not obtained, even with intensive alkali therapy in a stomach of low acid-secretory power. Acidity is lowered, but not abolished.



STARCH —
 BILE —
 MUCUS +
 BLOOD —

FJ 10 cc.

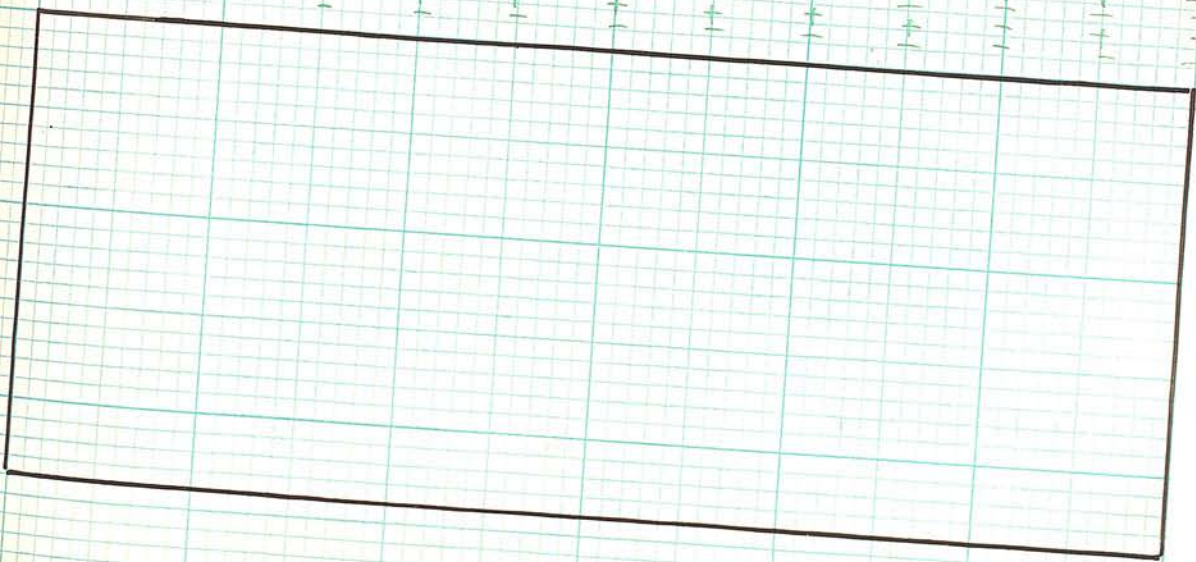
+	+	+	+	+	—
—	+	—	—	—	—
+	+	+	+	—	+
—	—	—	—	—	—



STARCH —
 BILE —
 MUCUS +
 BLOOD —

FJ 10 cc.

+	+	+	+	+	+	+	+	+	+	+	+
—	—	—	—	—	—	—	—	—	—	—	—
+	+	+	+	+	+	+	+	+	+	+	+
—	—	—	—	—	—	—	—	—	—	—	—



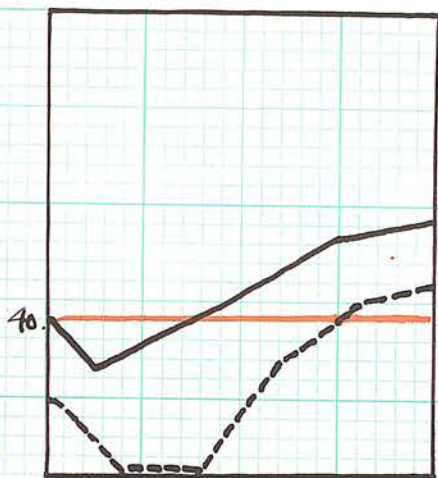
Control Y. 2.

Alexander I. (aet. 45). Machineman.

This man is well after an attack of peripheral neuritis (? secondary to specific central change) and his gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following the institution of a Sippy-alkali regime.

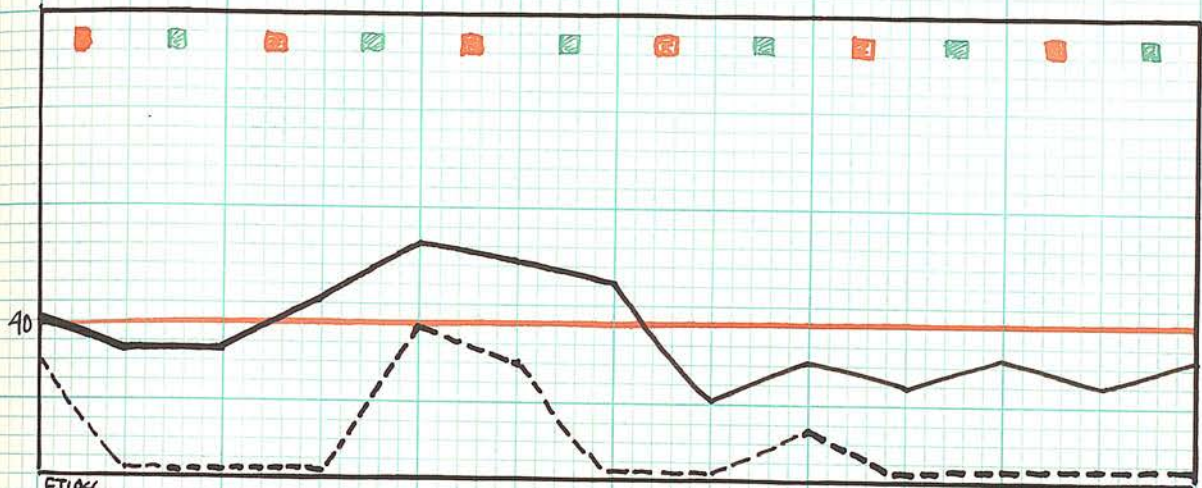
There is no history of any gastric trouble.

Notes: It will be seen that there is one considerable rise in acid in the long test meal here, this taking place half an hour after the administration of a dose of alkali, and being maintained for an hour: again demonstrating that achlorhydria is not obtained in the Sippy-alkali regime.



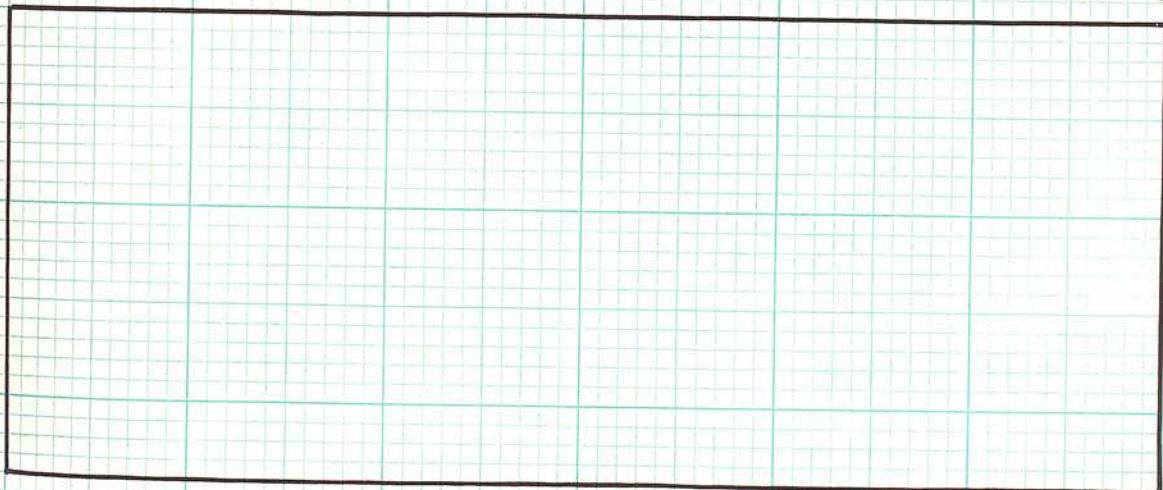
FJ 30cc.

STARCH	-	+	+	+	+	-
BILE	-	+	-	+	+	-
MUCOS	+	+	-	+	+	-
BLOOD	-	-	-	-	-	-



FJ 10cc.

STARCH	+	+	+	+	+	+	+	+	+	+	+	+	+
BILE	+	+	+	+	+	+	+	+	+	+	+	+	+
MUCOS	+	+	+	+	+	+	+	+	+	+	+	+	+
BLOOD	-	-	-	-	-	-	-	-	-	-	-	-	-



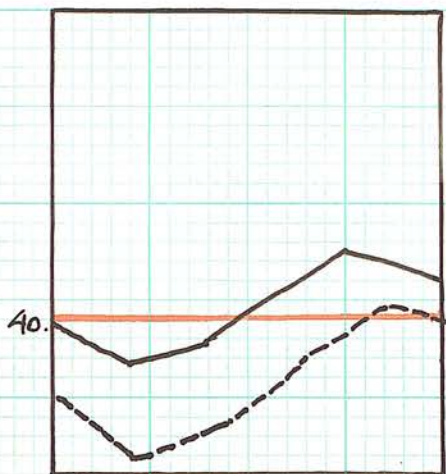
Control Y. 3.

Patrick McK. (aet. 36). Unemployed.

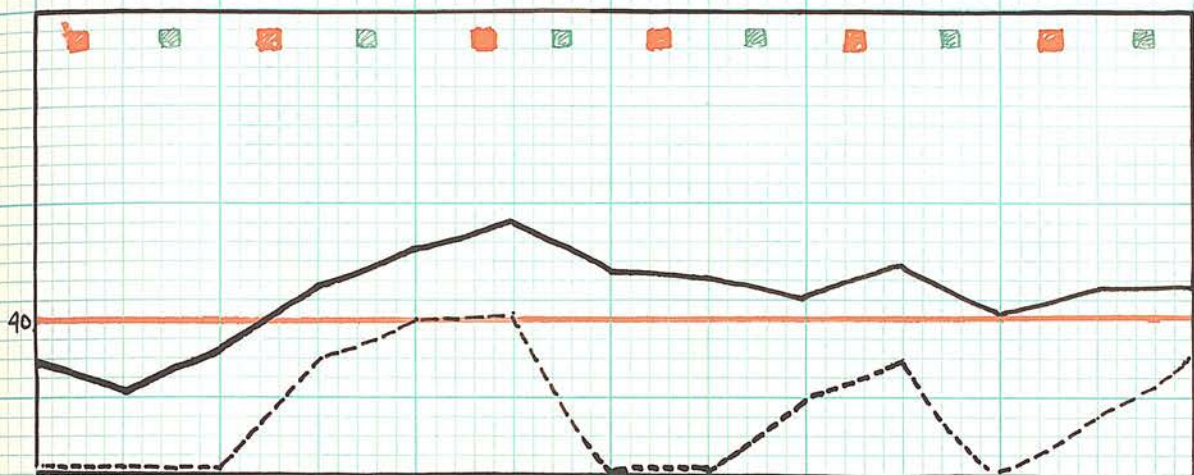
This man is under treatment for primary syphilis, but his gastric function is presumed normal, and was investigated by (1) ordinary test meal and (2) gastric analysis after institution of Sippy-alkali regime.

There is no history of any gastric trouble.

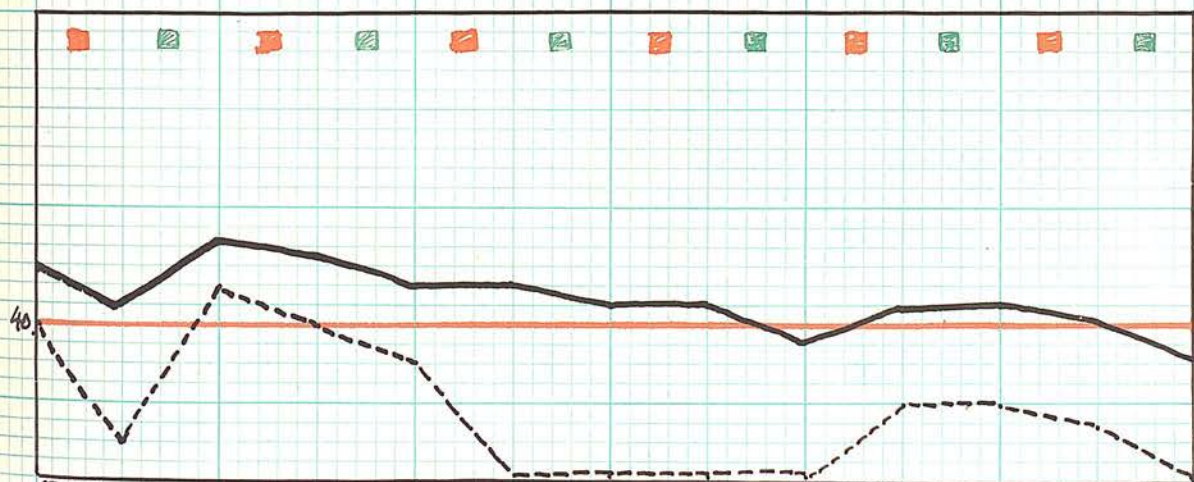
Notes: In this case it can be seen that a considerable rise in acidity can take place, even in the presence of intensive alkali therapy, though it may be noted that after a week there is considerable modification of this, and a lower general standard of solidity, but no sign of the development of constantly low acidity.



RT 10 cc.
 STARCH - + + + -
 BILE - - - - -
 MUCUS + + + + +
 BLOOD - - - - -



RT 10 cc.
 STARCH - + + + +
 BILE - - - - -
 MUCUS + + + + +
 BLOOD - - - - -



RT 25 cc.
 STARCH - + + + +
 BILE - - - - -
 MUCUS + + + + +
 BLOOD - - - - -

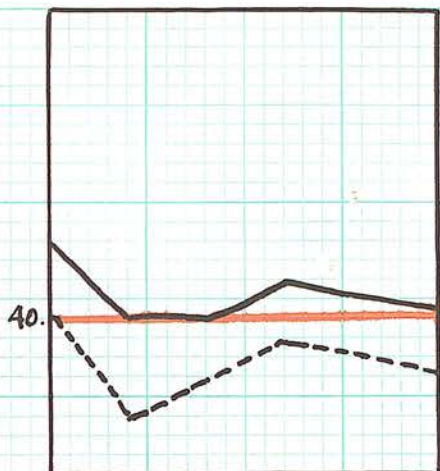
Control Y. 4.

John McF. (aet. 42). Clerk.

This man is convalescent after an auginal attack, and his gastric function is presumed normal. This case was investigated by (1) ordinary test meal and (2) gastric analysis following the institution of Sippy-alkali regime.

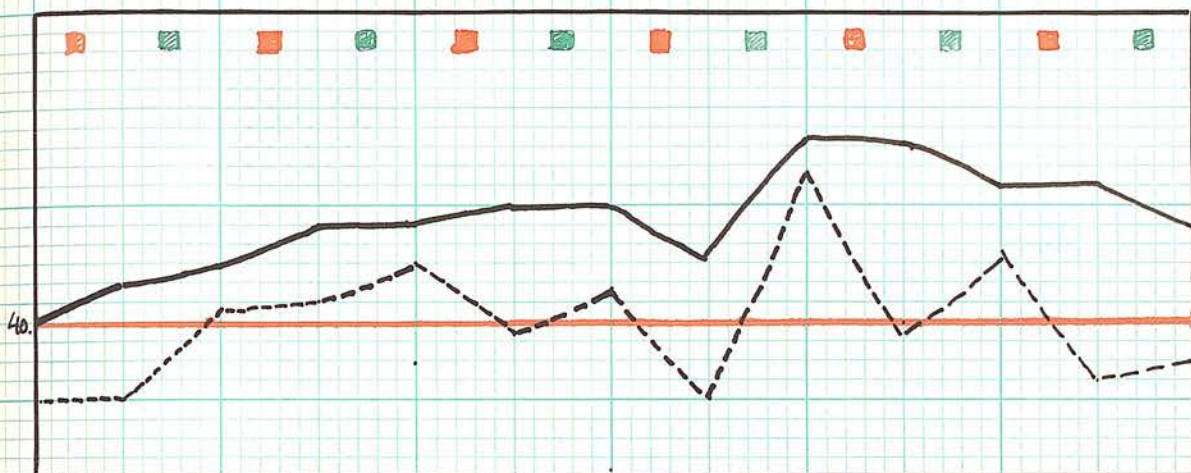
There is no history of any gastric trouble.

Notes: Marked hyperchlorhydria exists in this case, and it will be noted that this is profoundly influenced by the treatment, though achlorhydria is not obtained.

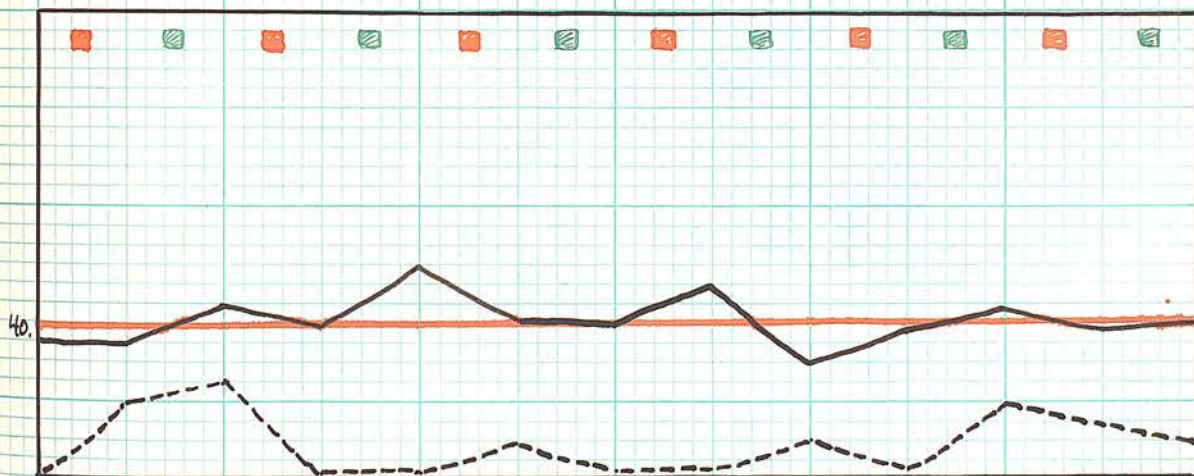


FJ 30 cc.

	1	2	3	4	5
STARCH	-	+	+	+	-
BILE	-	-	-	-	-
MUGGY	+	+	+	+	+
BLOOD	-	-	-	-	-



F87044

[illegible]

FT1044

[illegible]

Control Y. 5.

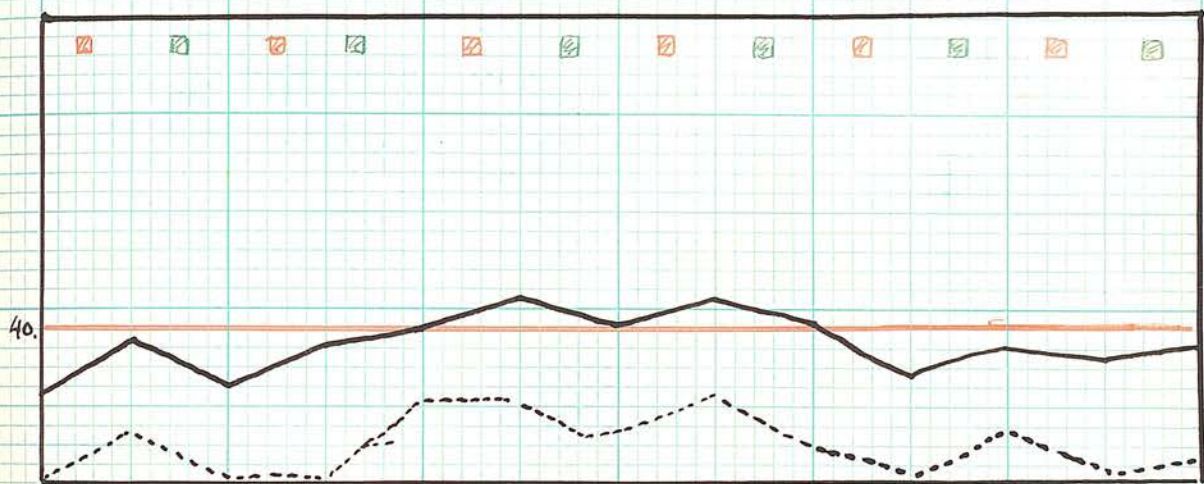
Janet McL. (aet. 28). Housewife.

This woman is well following an attack of broncho-pneumonia, and her gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following institution of Sippy-alkali regime.

There is no history of any gastric trouble.

Notes: It would appear in this case that the acid level is uninfluenced by the treatment: again achlorhydria is not obtained.

	FT 1065				
STARCH	-	+	+	+	+
WILE	+	-	-	-	-
MOSES	+	+	-	+	+
BLOOD	-	-	-	-	-

[illegible]

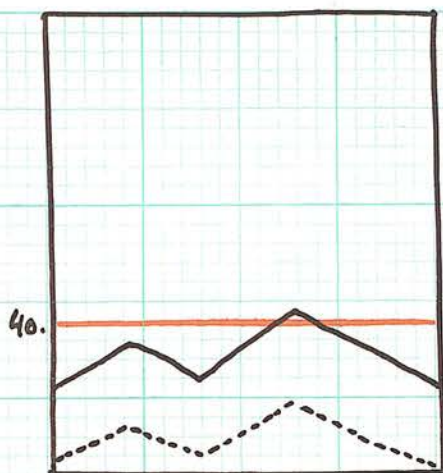
Control Z. 1.

James T. (aet. 42). Chemist.

This man is well following a coronary thrombosis, and his gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following the institution of Sippy-magsorbent regime.

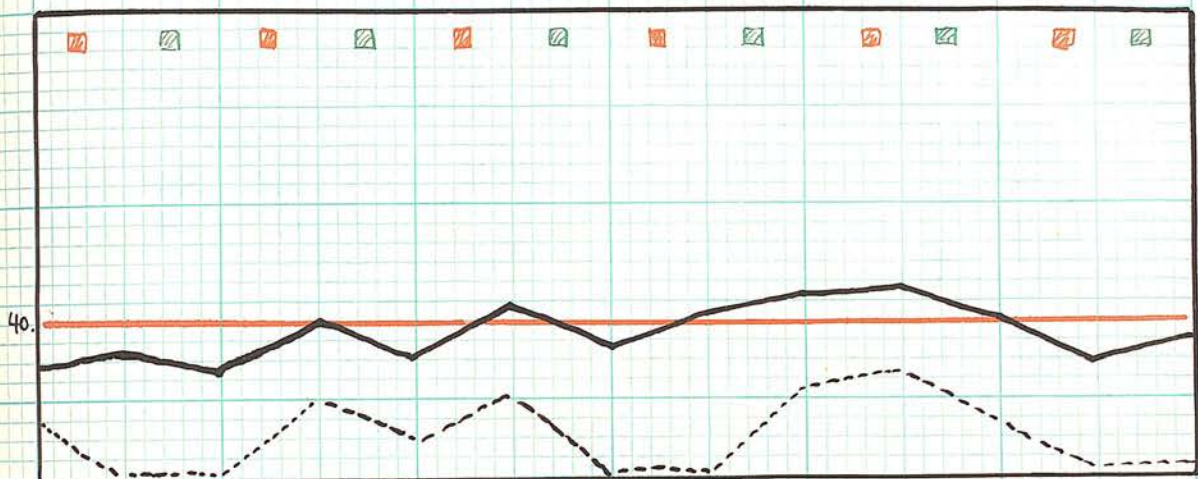
There is no history of gastric trouble.

Notes: The acid secretion in this case would appear to be influenced very little, if at all, by the treatment; no evidence is obtained that achlorhydria results from Magsorbent therapy.



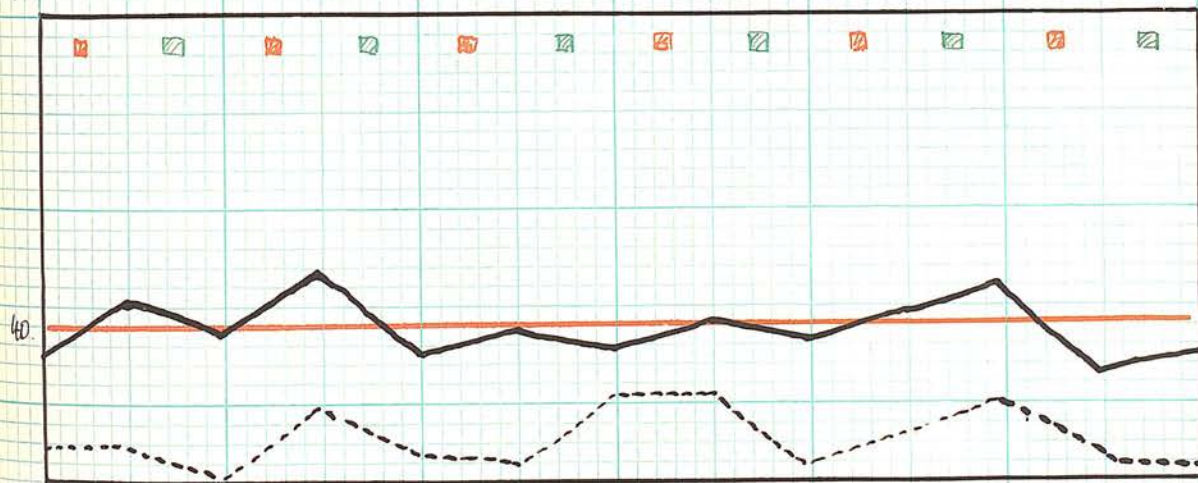
STARCH -
BILE +
MUCUS +
BLOOD -

87 10cc.



STARCH -
BILE +
MUCUS +
BLOOD -

87 20cc.



STARCH -
BILE +
MUCUS +
BLOOD -

87 10cc.

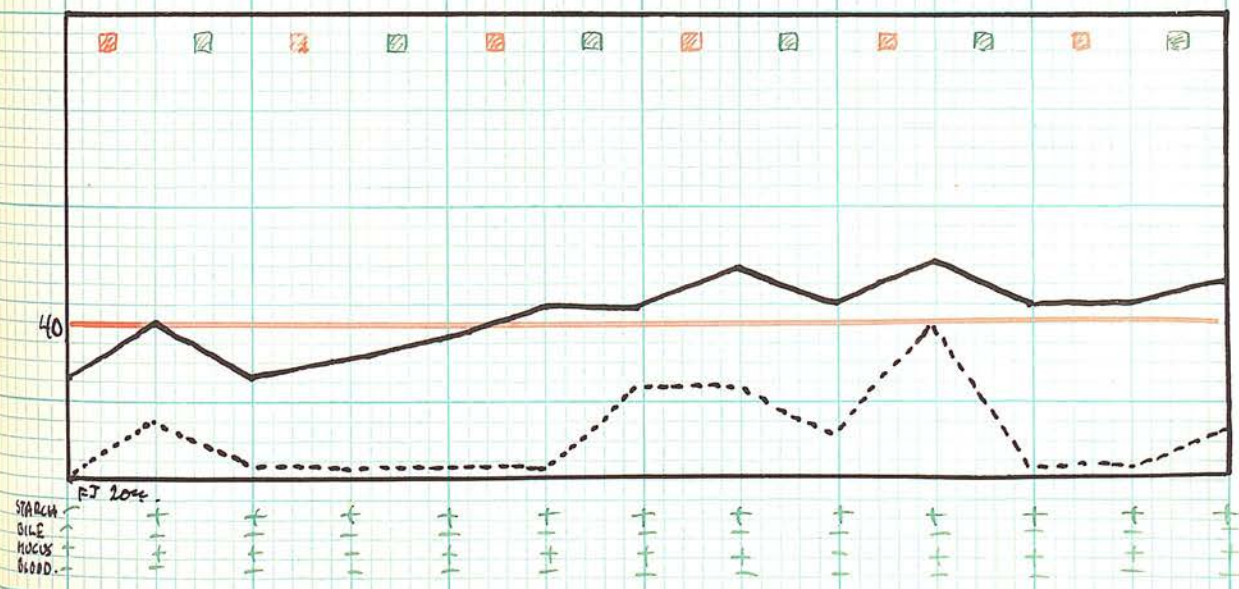
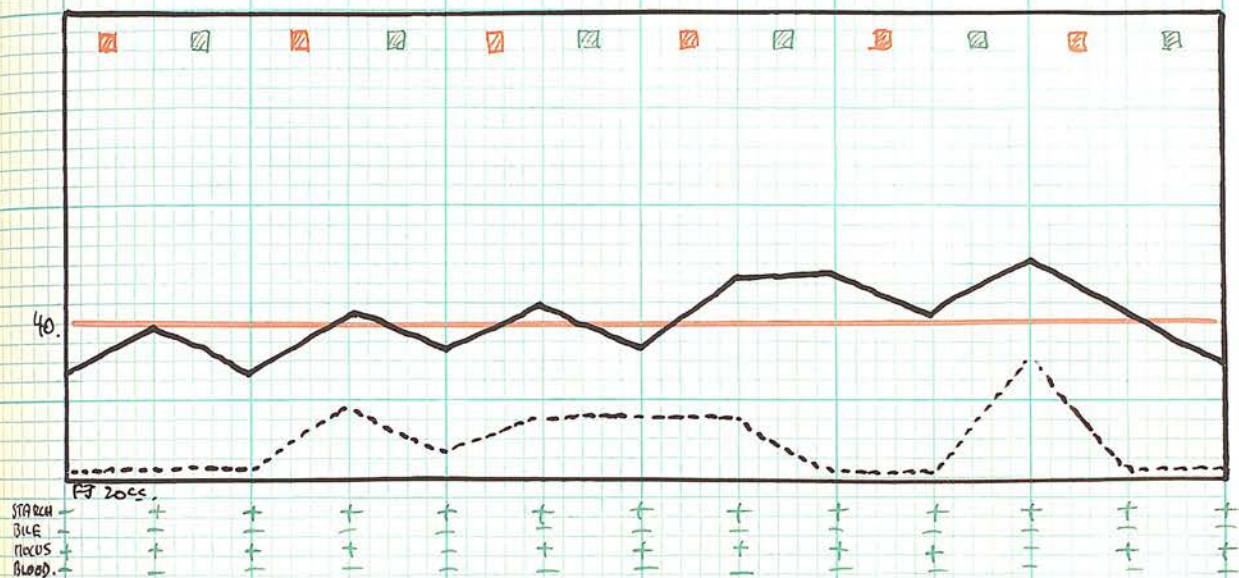
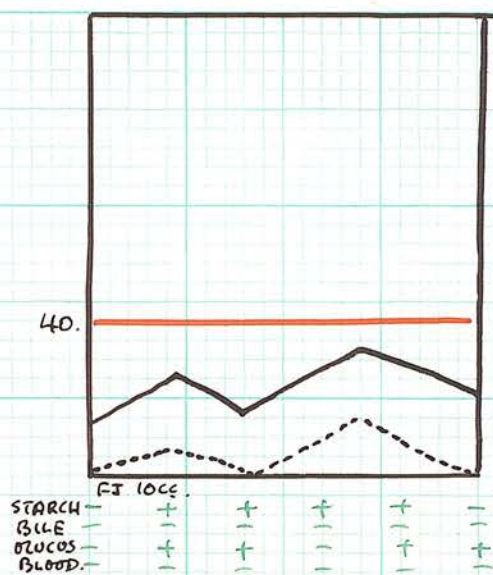
Control Z. 2.

Elizabeth F. (aet. 34). Draper's Assistant.

This woman is well, following an attack of pleurisy, and her gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following the institution of Sippy-magsorbent regime.

There is no history of previous gastric trouble.

Notes: Similar findings are obtained here as in the last case.



Control Z. 3.

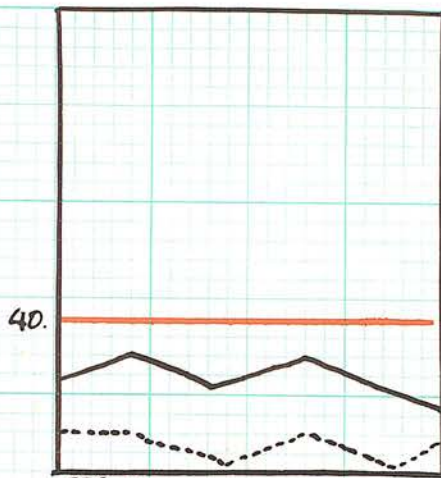
Alex. N. (aet. 22).

Miner.

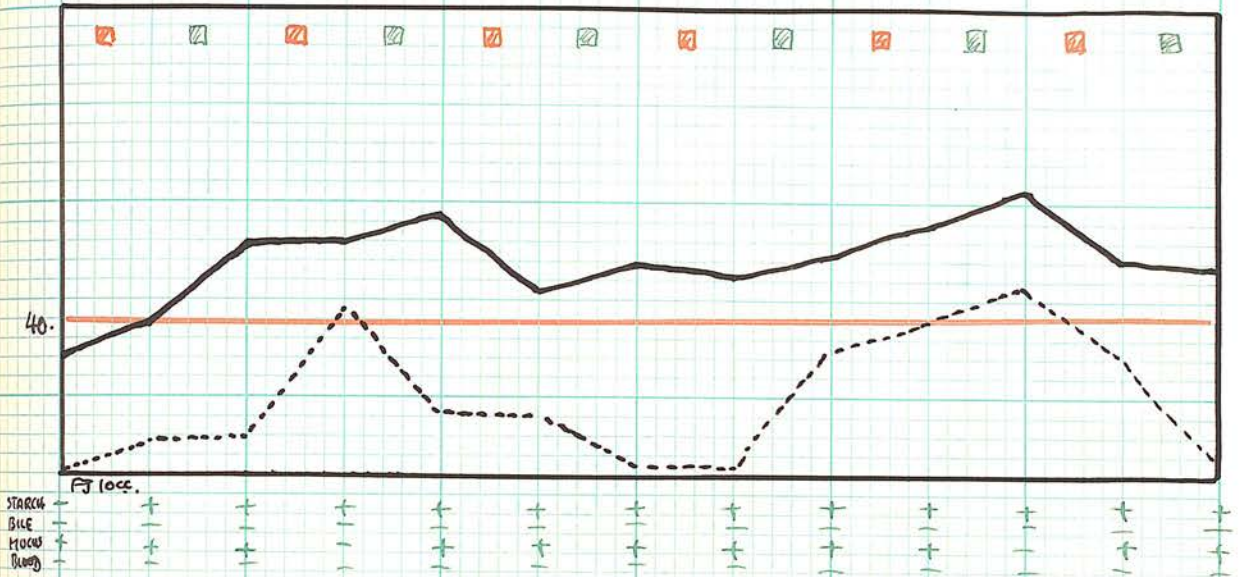
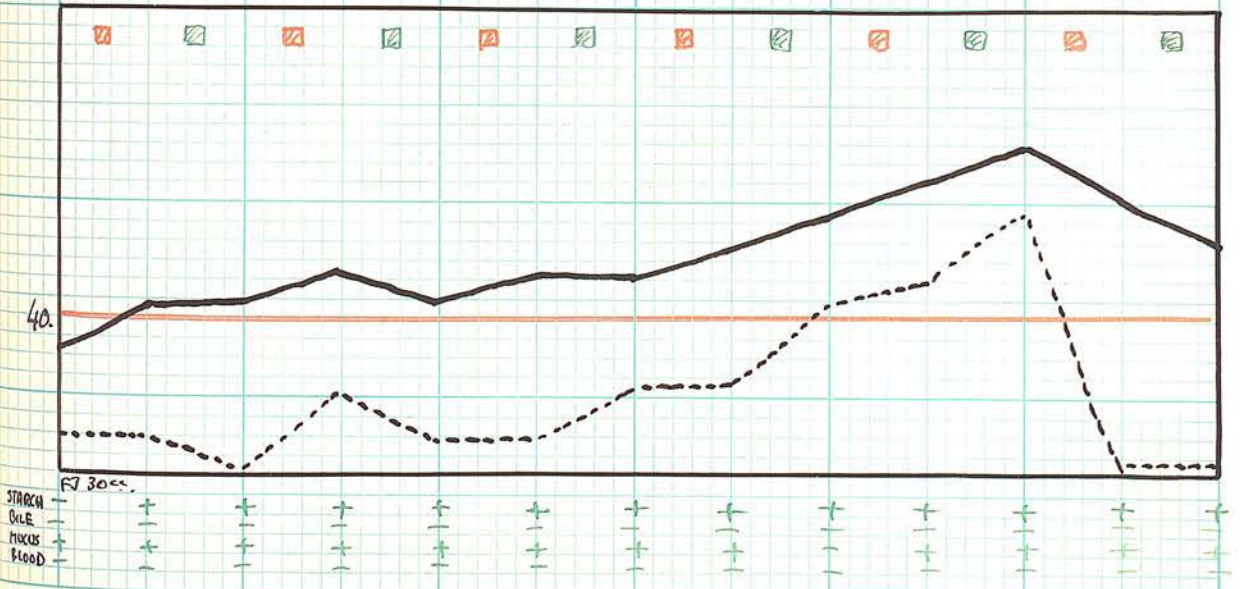
This man is well following acute lobar pneumonia, and his gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following institution of Sippy-Magsorbent regime.

There is no history of gastric trouble.

Notes: In the long test meals in this case there are surprising rises in acidity, apparently not affected by the therapy adopted: after one week there is very little difference.



	FJ 2005	FJ 2006	FJ 2007	FJ 2008	FJ 2009
STARCH	+	+	+	+	+
BILE	-	-	-	-	-
MUCUS	+	+	+	+	+
BLOOD	-	-	-	-	-

[illegible][illegible]

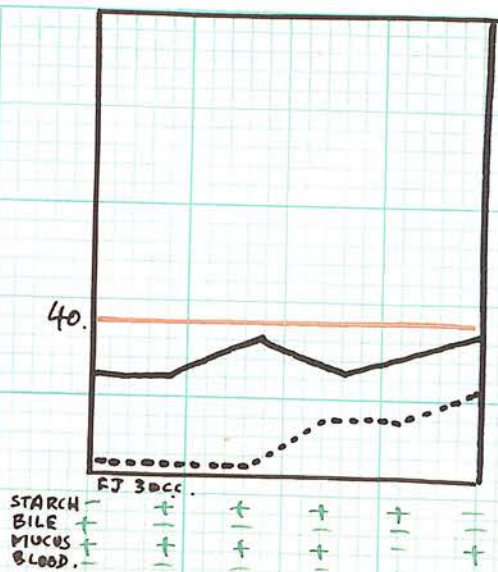
Control Z. 4.

James S. (aet. 44). Shop Assistant.

This man is well following an attack of acute rheumatism, and his gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following institution of Sippy-magsorbent regime.

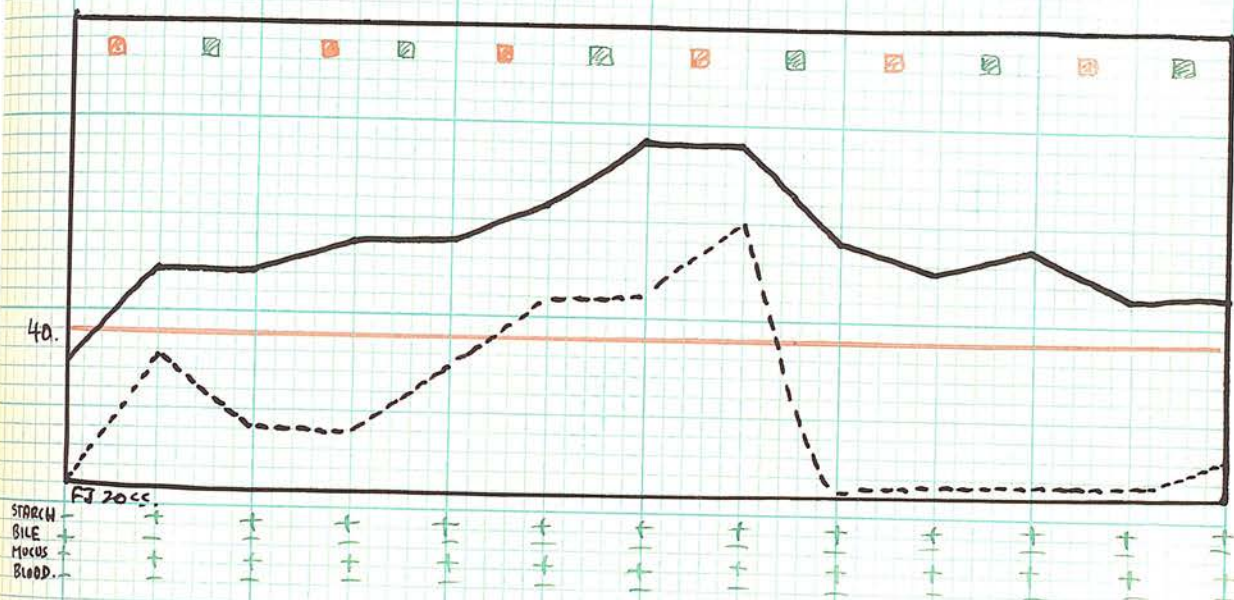
There is no history of gastric trouble.

Notes: It is again easily seen in this case that the treatment does not of necessity control acidity in the normal person.



FJ 30cc

STARCH
BILE
MUCUS
BLOOD.



FJ 20cc

STARCH
BILE
MUCUS
BLOOD.

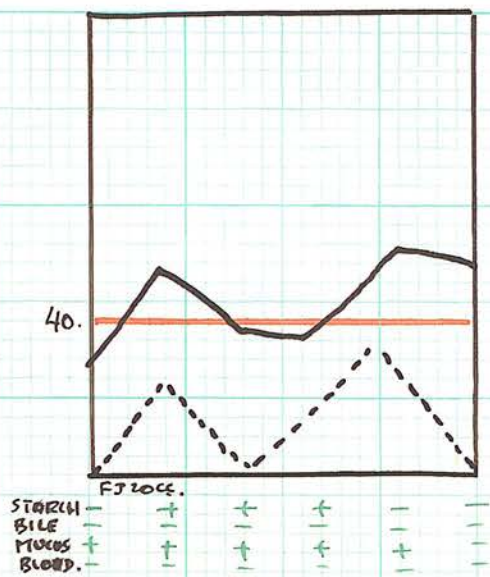
Control Z. 5.

Jean McS. (aet. 27). Domestic Servant.

This girl is well following an attack of pericarditis, and her gastric function is presumed normal. This was investigated by (1) ordinary test meal and (2) gastric analysis following institution of Sippy-magsorbent regime.

There is no history of gastric trouble.

Notes: In a normal person, as here, a very low acid level is not obtained by the therapy adopted.



CONSIDERATION OF CASES.

Forty-four cases were investigated, of which thirteen were presumed to have normal gastric function, and were included as control cases. Of the thirty-one cases exhibiting hyperchlorhydria, twenty-seven were suffering from duodenal ulceration, one from gastric ulceration and three from pylorospasm: (in two reflexly induced from chronic appendicitis: in one pylorospasm associated with a redundant duodenal loop.)

The following table indicates briefly the distribution of cases:-

<u>Treatment.</u>	<u>Controls.</u>	<u>Hyperchlorhydria Cases.</u>
Light Diet	3	5
Sippy-alkali	5	11
Sippy-Magsorbent	5	10
Surgical	-	5
	<u>13</u>	<u>31</u>

The method of investigation has already been referred to (vide ante).

Of these cases showing hyperchlorhydria, twenty-eight were male, and three female, a fact which would seem to suggest a definite sex incidence. As regards age, all these cases were aged between 20 and 50 years, and the vast majority were between 30 and 40 years, the average age being 36 years. The

diagnosis was confirmed radiologically in all except five cases, of which three were doubtful, and two showed no radiological abnormality. Clinical findings were accepted in the cases not confirmed by X-Ray, but it is worthy of note that the X-Ray findings were positive in 84% of cases, and in over 90% if the doubtful cases be included. The duration of symptoms varied considerably, in some cases being a few months, and in others many years: the average duration was five years. The universal symptom which obtained was epigastric pain or discomfort, coming on 1½-3 hours after food, and relieved by the taking of food or alkali: in the more long-standing cases this relief was frequently not obtained.

Eighty per cent. of the cases showed tenderness in the epigastrium, usually a little to the right of the midline, and sixty per cent. had some degree of muscular rigidity. In only fifty per cent., however, were there two signs elicited together. In ten per cent. of the cases "splashing" was elicited with a greater or less degree of ease. Acid eructation, flatulence and heartburn were constant findings, and constipation was usual: seven cases gave histories suggestive of attacks of haematemesis or melaena on some previous occasion. Most of the cases in which symptoms had been present

for a number of years admitted the occurrence of spells of remission of symptoms, having been free from time to time for periods lasting from one week to three months.

In almost all these cases the gastric lesion was the only one: a few, however, showed evidence of bronchitis, and three showed signs of mild endocrine disturbance, two being slightly hyperthyreoid, and one showing a slight degree of acromegaly.

In all cases, pain and discomfort subsided within the first two or three days following the institution of treatment, but tenderness and rigidity took rather longer to disappear, and were seldom gone before the fourth to the eighth day. Each case investigated has reported back to hospital once monthly since discharge, and their progress has been followed. There have been only two who have had mild relapses, and in both cases this was due to dietetic indiscretions, and was easily adjusted. The longest spell of supervision so far has been nine months, and this is too short a time to forecast the ultimate result of treatment. The immediate results, however, are most gratifying.

The behaviour of two cases demonstrated the possible dangers encountered: these cases were originally intended for inclusion in this series, but for obvious reasons were not included. The

first was that of a Jewess, with rather indefinite symptoms, and whose clinical condition appeared good; she "perforated" about two hours before a proposed test meal was started, and was operated on as a surgical emergency. In the male ward, a severe haematemesis occurred under rather similar circumstances. Neither catastrophe was predictable, and there was absolutely no sign of the imminence of such an occurrence.

It may be of interest to note here that a series of out-patients suffering from duodenal ulcers have been put on fourth week Sippy-alkali regime (see Appendix 1) without confinement to bed, and are all doing extremely well; symptom free, putting on weight, and carrying on at work.

DISCUSSION.

The introduction of the fractional test meal opened up a wide field for research into the behaviour of the stomach under varying conditions and in response to differing stimuli. One of the facts that has emerged from the flood of literature on gastric secretion is the remarkable prevalence of hyperchlorhydria and more specifically, the constancy of its association with peptic ulceration. This finding is borne out by the present investigation, in which it was noted that where peptic ulceration existed, hyperchlorhydria invariably co-existed. Theories advanced to explain this relationship have been discussed in a previous chapter.

Various modifications of the fractional test meal are in use today, and much valuable data has accumulated from analyses of the results of such meals. It will be recollected, however, that this test covers a spell of three hours only, and that the material used to stimulate secretion is not directly comparable to an ordinary meal such as the normal adult partakes of during the day. Hence some observers have attempted to examine specimens of gastric juice obtained over a longer period (Hellbrandt and Tepper 1936) varying from six to seventeen hours.

Diminution in the level of acidity as shown by the short test meal has been, in association with clinical improvement, one of the factors considered to be of importance in the healing of peptic ulcers, though it has been pointed out that this is not invariably true. Hurst (1929) noted that in thirty-two cases of duodenal ulceration, hyperchlorhydria became even more intense after healing of the ulcer by dietetic and neutralisation therapy than it had been during the acute phase; and Moore (1938) has expressed the same view, though he considers that the curve frequently becomes more normal in type after treatment, although the acidity is still excessive.

The disadvantages of the short test meal, however, are obvious; on physiological grounds the long test meal appears to give a better evaluation of gastric function. The patient is not so nervous and apprehensive, and by the end of two or three hours is used to the tube and able to proceed with his ordinary occupations of the day and to partake of his usual (hospital) meals. The "artificial" factor is thus to a large extent abolished. Hence this type of meal is useful in assessing the value of treatment, and it is this investigation that has been undertaken for the present thesis.

This method has not as yet been much applied, though Mann (1937) has reported an investigation of

the value of Magsorbent in this way, but only employing one case and no control case. Wosika and Emery (1936) used a modification of this method in an attempt to assess the utility of alkali and dried milk in peptic ulceration, but their cases did not include any exhibiting marked hyperchlorhydria, and the value of their results is uncertain.

For the purpose of the present thesis, certain criteria were employed. It has been assumed that an acidity of 40 represents the upper limit of normality: an acidity of above 40 represents hyperchlorhydria. The long test meal done immediately after the institution of treatment is compared directly with the shorter meal done a day or two previously, and the third test after two or three weeks treatment is considered with them. The case is considered to be under control if two criteria are satisfied.

- (a) The acidity does not rise above forty at any period in the twelve hours, and
- (b) The acidity rises above forty not oftener than twice in twelve hours, these not occurring in consecutive hours.

One modification is considered, that being that a case is partially controlled if the acidity exceeds forty three times, once being the fasting juice, and the three occasions not occurring consecutively. If these criteria are not satisfied,

the case is considered as not having been brought under control.

In three apparently normal persons investigated as controls, very different curves were obtained. One showed a very low acidity throughout the whole 12-hour period: this representing what one would anticipate from a normal person on normal diet. The second showed a similar curve, with one rise to above 40 during the day: this rise was considered as probably due to external factors, as it occurred two hours after swallowing the tube, and the woman was rather excitable and tending to retch at the time. There was no further rise when she became calmer. The third case, however, shows very marked hyperchlorhydria both in the short and in the long test meal, and is extremely difficult to explain: it is possible that she is naturally hyperchlorhydric and symptom free, or that she has a reflexly induced hyperchlorhydria. Actually, nothing was found to explain it: she was particularly stolid, and it seems unlikely that it is psychically induced. Her second test, though much more normal, cannot be classed definitely among the "expected" types of curve. It is thus apparent that normal persons may show a considerable variation: comparison may be drawn between this series and the normal cases used as direct controls on therapy.

In direct comparison with these cases, there may be considered the cases of peptic ulceration and hyperchlorhydria treated on light diet alone (Series 1). Although clinically those five patients improved rapidly, and became symptom free, no control whatever was obtained. The hyperchlorhydria remained after several weeks, and in one case, indeed, became more severe. The question arises here, then, whether or not the ulcer is healing in spite of the maintenance of excess acidity, and in view of the disappearance of the subjective and objective signs, it appears likely that healing is taking place. The complete rest and regular feeding is probably an important factor; as these cases have only been followed up so far for four to five months, it is as yet too early to give a definite pronouncement.

Another series of cases may be considered - Series Y. and Series 2, the latter being cases of hyperchlorhydria and peptic ulceration treated on strict Sippy-alkali regime. The former is a series of controls, presumably normal in gastric function, and investigated in an identical manner. It will be noted that satisfactory control is obtained in all the control cases, though frequent slight rises occur throughout the day. In no case is complete neutralisation effected, nor is a very low acidity maintained throughout the day without rises.

Sippy himself states that "control (i.e. absence of free HCl) should be maintained from the beginning of treatment" but this is not true, according to the present work, either in normal or abnormal cases. Of the cases of hyperchlorhydria treated thus, it will be seen that five are well controlled, two partially controlled, and four not controlled. Clinically, it was noted that there was no way of distinguishing between these cases: each case appeared to get on in a similar manner, and all became symptom free, felt well, and put on weight. Furthermore, in the cases not controlled within the first two days, no control was obtained after two to three weeks therapy, nor was there any special variation in their acid curves. It would appear that, if control is not obtained at once, no further improvement can be expected. The reasons for the establishment of control probably include the complete physical and physiological rest, the combining power of the food for acid, the ease with which soft foods are passed from the duodenum, and the rapid emptying of the stomach, increased by the alkali. One would expect that the late test meal would show a rise in acidity, due to the increased activity of the patient and the wider variety of food allowed, but this does not occur in the controlled cases. Whether control might have been

achieved in the uncontrolled cases by the use of greater amounts of alkali was not investigated, as the dosage of alkali was constant.

It is worthy of note that in these cases, on massive alkali therapy, there was no case of alkalosis developing. This complication was noted by Hardt and Rivers (1923) to occur in some of Sippy's own cases, and has since been discussed by Oakley (1935). In the cases under consideration, however, no definite signs developed. The alkali reserve and the blood urea tended to rise a little during treatment, but they were always well within normal limits. No clinical signs suggestive of alkalosis were noted, though this was carefully watched for; the urine, though alkaline, never contained albumen or casts. It is interesting, when considering the possible dangers of alkali therapy, to note that Watchford (1938) has pointed out that sodium bicarbonate, as well as possessing other well known and undesirable side-effects, is highly pleionectic to haemoglobin (Barcroft), and may induce mild anoxaemia. It may be concluded that the Sippy-alkali treatment is not always successful from a biochemical standpoint.

Another series of cases was treated on a similar regime, with the substitution of Magsorbent for alkali, namely, Series Z, presumably normal controls on this regime, and Series 3, cases of

hyperchlorhydria and peptic ulceration on Sippy-Magsorbent regime. One drachm of magsorbent (a proprietary preparation of Magnesium Trisilicate) was given in place of a dose of alkali, and the diet remained the same. Of the normal cases, three of the five were not satisfactorily "controlled" on this therapy, as compared with a similar series well controlled on Sippy-alkali. Of the hyperchlorhydric cases, however, eight were controlled and two not controlled - definitely a better control than the analogous series on alkali. This may be due to the absorbent powers and prolonged action of the drug, but another factor must be considered, namely, that the cases treated do not exhibit primarily such a high degree of hyperchlorhydria as the other series. Further, in general the duration of symptoms is less, and it is possible that the ulcers were at a less intractable stage of chronicity. However, it is to be noted here again that complete neutralisation is not effected, either in the normal or the abnormal case. Here, too, it was found impossible to distinguish on clinical grounds between cases controlled and those not controlled, and it was noted that if control was not achieved at once, no further benefit resulted. It would appear that a long test meal on the first or second day following the institution of treatment will give all the necessary information

concerning control or lack of control. One case developed severe diarrhoea when having Magsorbent, and the dose had to be slightly reduced: almost all the others complained of constipation, and required mild aperients. Another factor arises in connection with the consideration of the whole series - 2 and 3 - together, and that is that it is in the older persons and those in whom symptoms have been present for a long time who are most difficult to control. It is well known that with advancing age, gastric secretion diminishes, and it is possible that abnormal stimulation may therefore give rise to a more "malignant" type of hypersecretion in the older person than in the more resilient and easier adjusted young one. It is obvious, too, that a chronic ulcer, with inflammatory and fibrous change around it, is much more difficult to heal than an early one without these degenerative and regenerative processes, and the probability of some degree of pyloric narrowing and hence poorer drainage is of importance in the chronic case. (Improvement in drainage is obviously one of the ways of lowering acidity - in pyloric obstruction the acid is not neutralised, as the acid does not leave the stomach, and the duodenal contents do not enter it).

Of the surgical cases not a great deal need be said. They were included in order to demonstrate the type of curve found in cases of ulcer verified at

operation, and agree with the other cases of ulcer verified clinically and radiologically. It is of interest, however, to note that the acidity does not drop at once after a gastro-enterostomy has been performed, but may take a few weeks or even months to diminish. Mailer (personal communication) in collaboration with Reid at the Mayo Clinic, considers that the acidity probably rises again after six months or a year, and gradually attains its pre-operative level.

SYNOPSIS AND CONCLUSIONS.

A series of cases of peptic ulceration were investigated, all of which exhibited hyperchlorhydria, which was more marked in some than in others. In all cases ulceration was clinically present, and in most this was confirmed radiologically: in a very small number it was confirmed surgically. These cases were treated on a Sippy regime, and it was noted that the hyperchlorhydria was modified by this treatment, but seldom entirely abolished. The adjuvants to dietetic treatment - alkalies or Magsorbent - showed little specific difference in their effects, though with the former there was a tendency for the alkali reserve of the blood to rise. The hyperchlorhydria was not affected by light diet without the use of drugs, though symptoms appeared to subside as rapidly. Biochemically, the control achieved by the Sippy treatment was not perfect, and in no case was complete neutralisation of acidity effected; the control did not become more perfect as treatment proceeded, though clinically improvement was satisfactory. It was found, however, that in normal individuals on a similar regime, the same state of affairs existed to a lesser degree, and that neutralisation was not effected, even in the case with a comparatively low initial acidity. Clinically, it was found that no distinction could be drawn between cases "controlled" or "not controlled" as judged by biochemical findings.

CONCLUSIONS.

- (1) Cases of duodenal ulcer exhibit hyperchlorhydria.
- (2) In at least 84% of cases the ulcer is demonstrable radiologically.
- (3) During treatment, the subjective signs disappear within the first two or three days, and the objective signs within the first ten days in cases responding to treatment.
- (4) The hyperchlorhydria is hardly modified at all by light diet without drugs, but symptoms are relieved.
- (5) The hyperchlorhydria is markedly modified by a Sippy regime, and is better controlled by Magnesium Trisilicate than by alkalies in association with this diet.
- (6) Control, as judged by gastric analysis, is not perfect either in the normal or the hyperchlorhydric case.
- (7) Control does not become more perfect during treatment, as judged by successive tests; and hence, degree of control can be assessed by tests within the first few days following the institution of treatment.
- (8) During treatment with massive dosage of alkali the risks of alkalosis developing are so slight as to be negligible.

- (9) No distinction can be drawn on clinical grounds between cases in which the hyperchlorhydria, during treatment, is controlled at once, is controlled after a few weeks, or is not controlled at all.
- (10) The "long test meal" (lasting at least 12 hours) is of much greater value than the ordinary short test meal, in the investigation of gastric function, and the assessment of the effect of different agents upon secretion.

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APPENDIX I.Diet adopted.1st and 2nd Days.

1 pint milk)	required
1 pint single cream		daily.

Each feed will consist of $2\frac{1}{2}$ ozs. milk and $2\frac{1}{2}$ ozs. cream = total 1 teacupful.

6 a.m.	Cream and milk.
7 a.m.	Powder No. 1.
8 a.m.	Cream and milk.
9 a.m.	Powder No. 2.
10 a.m.	Cream and milk.
11 a.m.	Powder No. 1.
12 noon	Cream and milk.
1 p.m.	Powder No. 2.
2 p.m.	Cream and milk.
3 p.m.	Powder No. 1.
4 p.m.	Cream and milk.
5 p.m.	Powder No. 2.
6 p.m.	Cream and milk.
7 p.m.	Powder No. 1.
8 p.m.	Cream and milk.
9 p.m.	Powder No. 2.
10 p.m.	Powder No. 1.

3rd, 4th and 5th Days.

$1\frac{1}{2}$ pints milk)
 $\frac{3}{4}$ pint single cream) required
) daily.

Each feed will consist of $2\frac{1}{2}$ ozs. milk and $2\frac{1}{2}$ ozs. cream (total 1 teacupful) unless otherwise stated.

6 a.m. Cream and milk.
 7 a.m. Powder No. 1.
 8 a.m. Milk food made with and served with
 cream, total 1 teacupful.
 9 a.m. Powder No. 2.
 10 a.m. Cream and milk.
 11 a.m. Powder No. 1.
 12 noon 2 tea rusks soaked in hot milk and
 served with cream or milk food
 as at 8 a.m.
 1 p.m. Powder No. 2.
 2 p.m. Cream and milk.
 3 p.m. Powder No. 1.
 4 p.m. Switched egg, cream and milk
 1 teacupful.
 5 p.m. Powder No. 2.
 6 p.m. Cream and milk.
 7 p.m. Powder No. 1.
 8 p.m. Milk food as at 8 a.m.
 9 p.m. Powder No. 2.
 10 p.m. Powder No. 1.

6th, 7th and 8th Days.

2 pints milk)
) required
 $\frac{3}{4}$ pint single cream) daily.

1 oz. i.e. 2 tablespoonfuls.

If constipated, take Powder No. 1 in place of No. 2
 as often as necessary.

If diarrhoea occurs, take Powder No. 2, in place
 of No. 1 as required.

- 6 a.m. Cream and milk 1 teacupful (5 ozs.)
- 7 a.m. Powder No. 1.
- 8 a.m. Cream and milk with 2 tea rusks, or
 milk food 7 ozs. 2 tablespoonfuls
 strained orange juice diluted in
 water.
- 9 a.m. Powder No. 2.
- 10 a.m. Switched egg with warm milk and a
 little sugar - 7 ozs.
- 11 a.m. Powder No. 1.
- 12 noon Cream and milk 1 teacupful. Milk
 pudding 1 large saucerful (no
 whole rice). 2 tablespoonfuls
 strained orange juice, diluted
 with water.
- 1 p.m. Powder No. 2.
- 2 p.m. Cream and milk 1 teacupful (5 ozs.)
- 3 p.m. Powder No. 1.
- 4 p.m. Egg custard 6 ozs. served with cream.
 2 tablespoonfuls strained orange
 juice diluted with water.
- 5 p.m. Powder No. 2.
- 6 p.m. Cream and milk 1 teacupful (5 ozs.)

- 7 p.m. Powaer No. 1.
- 8 p.m. Bengers Food or other milk food
8 ozs.
- 9 p.m. Powder No. 2.
- 10 p.m. Powder No. 1.

9th to 14th Days.

2 pints milk)
) required
 $\frac{3}{4}$ pint single cream) daily.

1 oz. i.e. 2 tablespoonfuls.

If constipated, take Powder No. 1 in place of No. 2 as often as necessary.

If diarrhoea occurs, take Powder No. 2 in place of No. 1 as required.

- 6 a.m. Cream and milk 1 teacupful (5 ozs.)
- 7 a.m. Powder No. 1.
- 8 a.m. Cream and milk 5 ozs. 1 slice
 crisp toast, buttered when cold,
 or 3 rusks with butter, or
 strained porridge 1 teacupful
 with cream. Orange juice
 2 tablespoonfuls.
- 9 a.m. Powder No. 2.
- 10 a.m. Cream and milk and switched egg -
 total $\frac{1}{2}$ pint.
- 11 a.m. Powder No. 1.
- 12 noon Milk pudding 1 large saucerful
 (no whole rice). Cream and milk
 1 teacupful. Orange juice
 2 tablespoonfuls.
- 1 p.m. Powder No. 2.
- 2 p.m. Cream and milk 8 ozs.
- 3 p.m. Powder No. 1.
- 4 p.m. As at 12 noon - any milk food
 $\frac{1}{2}$ pint.
- 5 p.m. Powder No. 2.

- 6 p.m. Egg custard 6 ozs.
Cream and milk 5 ozs.
Orange juice 2 tablespoonfuls.
- 7 p.m. Powder No. 1.
- 8 p.m. Cream and milk, or milk food
 $\frac{1}{2}$ pint.
- 9 p.m. Powder No. 1.
- 10 p.m. Powder No. 2.

3rd Week.

2 pints milk)
 $\frac{1}{2}$ pint single cream) required
) daily.

1 oz. i.e. 2 tablespoonfuls.

If constipated, take Powder No. 1 in place of
 No. 2 as often as necessary.

If diarrhoea occurs, take Powder No. 2 in place of
 No. 1 as required.

- 6 a.m. Cream and milk 1 teacupful.
- 7 a.m. Powder No. 1.
- 8 a.m. 1 cup very weak tea freshly made -
 serve with cream. Milk food or
 strained porridge 1 teacupful with
 cream. $\frac{1}{2}$ slice toast - buttered
 when cold. Orange juice 4 table-
 spoonfuls.
- 9 a.m. Powder No. 2.
- 10 a.m. Switched egg in milk with cream,
 $\frac{1}{2}$ pint.
- 11 a.m. Powder No. 1.
- 12 noon Steamed fish 4 ozs. with butter or
 white sauce. Curds or 1 saucerful
 of milk pudding with cream (not
 whole rice). $\frac{1}{2}$ slice toast -
 buttered when cold. Orange juice
 4 tablespoonfuls - may be added
 to jelly, or prune or apple
 sieved as on recipe.
- 1 p.m. Powder No. 1.
- 2 p.m. Cream and milk, $\frac{1}{2}$ pint.
- 3 p.m. Powder No. 1.
- 4 p.m. Lightly boiled egg. 1 slice toast,
 buttered when cold, or thin bread
 and butter. 1 cup very weak tea,
 freshly made - served with cream.

- 5 p.m. Powder No. 2.
- 6 p.m. Cream and milk, $\frac{1}{2}$ pint.
- 7 p.m. Powder No. 1.
- 8 p.m. Milk pudding or custard or milk food.
Orange juice 2 tablespoonfuls.
- 9 p.m. Powder No. 2.

4th Week.

- 7.30 a.m. Powder No. 1.
- 8 a.m. BREAKFAST - 1 cup weak tea with cream or milk. 1 teacup strained porridge with cream or milk.
- 1 egg boiled or poached or scrambled if desired.
- 1 slice crisp toast - buttered when cold. Orange juice, 2 tablespoons.
- 9 a.m. Powder No. 2.
- 10.30 a.m. Cream and Milk, $\frac{1}{2}$ pint.
- 11.30 a.m. Powder No. 1.
- 12.30 p.m. DINNER - Fish, chicken, tripe, rabbit, calf's head, sheep's head, fresh minced meat (omit if this gives pain).
- Vegetable passed through sieve.
- Curds or saucer of milk pudding with cream (no whole rice).
- Always baked or stewed apple or fruit as on recipe.
- A tablespoonful potato mashed or baked in skin.
- 1.30 p.m. Powder No. 2.
- 3 p.m. Cream and milk 5 ozs. (1 teacup).
- 5 - 6 p.m. TEA - 1 cup weak tea with cream. Egg or fish - not fried now or at supper.
- 1 slice bread and butter, 2 tea rusks or slice toast. Sometimes a small piece sponge cake.
- Orange juice 2 tablespoonfuls.

- 6 p.m. Powder No. 2.
- 8 - 9 p.m. SUPPER - Milk pudding, or any milk food served with cream; total amount 1 breakfast-cupful.
2 plain biscuits or rusks.
- 9 p.m. Powder No. 1.
- 10 p.m. Powder No. 2.

Not allowed - soups, onions, fried foods, highly seasoned foods such as sausages, twice cooked meats, stews, coarse vegetables, new bread, scones, rolls, pastry, alcohol, strong tea, coffee. No pips, skins, or seeds of fruit are allowed.